Reprinted from THE PRACTITIONER.]

HEART-BEAT AND PULSE-WAVE.

BY C. S. ROY, M.D., F.R.S.,

Professor of Pathology,

AND J. G. ADAMI, M.A., M.B.,

Demonstrator of Pathology, in the Univeriity of Cambridge.

[From the *Cambridge Pathological Laboratory.*]

THE clinical importance of an exact understanding of the meaning of graphic records of the heart and pulse, together with the fact that the graphic method is the most convenient for investigating certain physiological and pathological questions connected with the circulatory system, have induced us from time to time to make a few observations on the matters expressed by the title of our paper. In these observations we have employed various new instruments, which must be described in order that our tracings may be comprehended.

We will try, however, to say as little about the methods as is compatible with giving a reasonably intelligible account of our results. These results, we believe, are of a kind which will be found of value at the bedside, although to make them clear it is

necessary for us to refer to some matters which at first sight may seem to be of interest to the physiologist only.

It will be most convenient to consider the heart-beat first, for the simple reason that the characteristics of the pulse-wave are due for the most part to the manner in which the ventricular contraction takes place.

Graphic records of the heart-beat may be obtained in a variety of ways. The contraction and expansion of the muscular wall of the ventricle or auricle, for example, may be recorded. It is possible, also, to obtain a curve of the changes in the intra-ventricular pressure, as was done by Chauveau, Marey, and others, and more recently and accurately by Rolleston. The changes in the diameter of the ventricles, both in the antero-posterior and in the transverse direction, may be graphically determined: and the movements of the apex can in like manner be investigated. We may obtain curves of the contraction and expansion of the *musculi papillares*, showing the movements of the free edges of the auriculo-ventricular valves. Or again we may register the changes in volume of the heart as a whole. The kinds of tracings which we have just mentioned are perhaps among the most important of those by which the characters of the heart-beat can be studied, although there are many others which need not here be referred to.

SECTION I.

CONTRACTION-CURVE OF THE VENTRICULAR WALL.

Let us consider first of all the curve of contraction and expansion of the heart-muscle forming the ventricular wall. In order to obtain trustworthy tracings we must employ a method which will not hinder the movements of the heart as a whole. The method must also give tracings which will not be affected by these movements. The 'myocardiograph' (as we may call it to distinguish it from other forms of cardiograph) shown somewhat diagrammatically in Fig. 1, fulfils these conditions. By its means it is easy to obtain trustworthy graphic records of the variations in the distance apart of any two points on the surface of the heart-wall. Its construction is as follows: The light

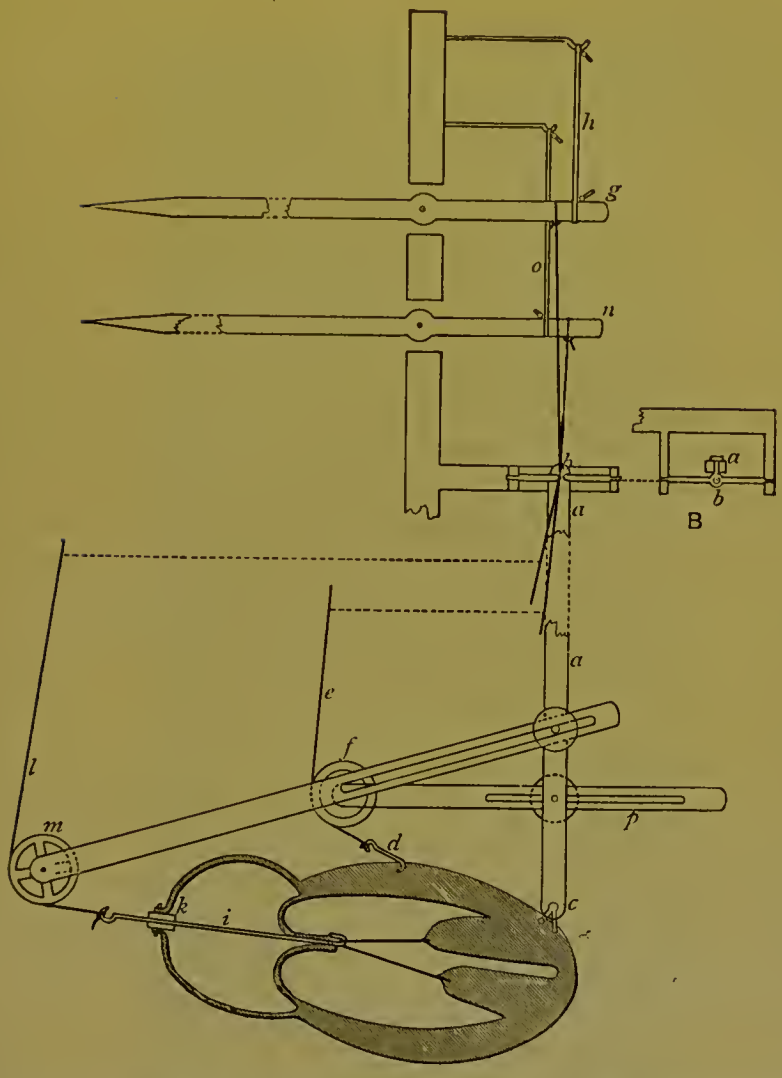

FIG. 1.—Myocardiograph for mammalian heart shown semi-diagrammatically. The light vertical rod *a*, which for convenience of space is shown shortened in the figure, is slung from the pivots which are represented in section as seen from above in B. This arrangement allows the rod *a* to swing freely, the centre of rotation being the small hole at *b* (in B). The lower end, *c*, of this rod is fixed to the surface of the heart-wall as seen in the figure. To obtain tracings of the heart-wall, the small hook *d* is inserted in the visceral pericardium at a convenient distance from the end of the rod *a*. To this hook is attached a strong silk thread *e*, which after passing round the light grooved pulley *f* is conveyed upwards through the small hole *b* to the lever *g*, being kept taut by the fine rubber thread *h*.

To obtain tracings of the contraction of the *musculi papillares*, the fine hooked wire *i* is inserted through the auricular wall and hooked over one of the mitral flaps. It slides easily in the collar *k*, which is tied to the edges of the opening in the auricular wall. To this is attached the thread *l*, which after passing round the light pulley *m* is conveyed upwards through the hole *b* to the lever *n*, being kept taut by the rubber thread *o*.

wooden rod (*a*) is slung on pivots or gimbals (*b*), somewhat after the manner of the ordinary mercurial barometer used on board ship. The lower end (*c*) of the rod is fixed to the heart-wall at any desired point by means of a thread which has been passed under a fold of the visceral pericardium. The end of the rod, from the manner in which it is slung, can follow the complicated movements of the heart without hindering them. Projecting from the rod near its lower end is thehorizontal arm (*p*), which carries at its extremity a light vulcanite grooved pulley (*f*). Round this latter runs a strong silk thread (*e*), which is attached to a minute metal hook (*d*) fixed in the ventricular wall at any desired distance from the end of the rod (*a*). After passing round the pulley, the thread is carried upwards through a small hole (*b*) placed at the centre of rotation of the rod, as can be seen from the sketch elevation of the pivoting arrangement B in Fig. 1. From this hole the thread goes upwards to the recording lever (*g*), being kept taut by a fine indiarubber spring (*h*). The object of passing the thread through the hole at the centre of rotation of the rod is to allow free movement of the end of the latter with the heart, to which it is fixed, without any pull on the recording lever being thereby produced. The position of the lever is altered only by alterations in the distance between the point where the hook is fixed, and the point to which the rod is tied. It need hardly be added that in order to make use of this instrument the heart must be exposed by making a "window" in the thorax, the animal (a dog in our experiments) being curarised as well as anæsthetised, and the respiration being, of course, carried on artificially.

On the myographic curve of the auricular wall we do not propose to say anything here. The curves which are represented below in this section of our paper were obtained from the wall of the left ventricle, although, as they differ in no essential particular from curves obtained by the same method from the right ventricle, it must be understood that what we have to say about them applies equally to the two ventricles.

The myographic curve from the ventricular wall varies somewhat in character according to the relative position of the two points to which the instrument is attached. Let us consider first the curve given by this method when the two points

lie in a line running from base to apex, and more or less parallel

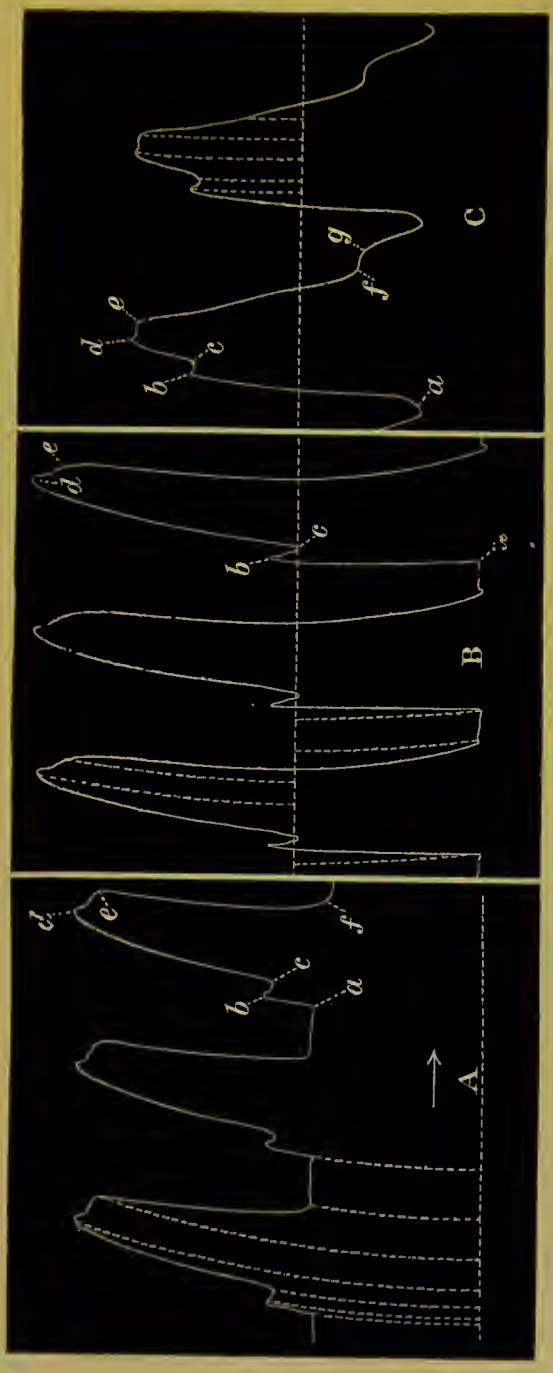

Fig. 2.

therefore with the interventricular sulcus. This, for convenience sake, we may call the "longitudinal curve" of the

ventricular wall, to distinguish it from that obtained from a part lying between two points on any line running round the ventricles, parallel to the auriculo-ventricular sulcus, which may similarly be termed the " transverse ventricular " curve.

The curves (A, B, C) shown in Fig. 2 are examples of tracings registering the contraction between two points in the middle third of a line joining the base and apex of the left ventricle at some distance from the ventricular septum. The illustration shows three different tracings, in all of which a rise of the recording lever corresponds to contraction of the ventricle wall, and a descent to expansion. The contraction begins, then, in each at *a*. It can be seen that the heart-wall contracts at first rapidly, until the shortening is suddenly arrested at the point *b*, the height of which on the ascending line varies under different conditions. In curve A (Fig. 2), it is low down, near the commencement of the ascent; in curve B it is about half-way up; while in curve C it is near the top. This arrest of the contraction is often followed by a certain degree of expansion, producing a more or less well-marked notch at *c*. On the other hand, it may (as in Fig. 4, A) be followed by a simple cessation of the contraction, or even by mere slowing, though this, according to our experience, is unusual. After the notch at *c*, the contraction continues, but is more slow than before. What is the cause of this interruption of the shortening? We at first thought that it must be due to the tightening of the auriculo-ventricular valves as a result of the rise of the intra-ventricular pressure, produced by the contraction of the heart-wall. That it results from something more than this will be seen when we come to compare the curve of contraction of the *musculi papillares* with that of the heart-wall itself.

We need only remark here that the time between the commencement of the contraction of the heart-wall and this break in the shortening varies under varying conditions, all of which however affect the amount of blood contained within the ventricle at the commencement of systole. The greater the expansion of the heart in diastole, the sooner does this break in the ascending line of the contraction curve follow its commencement, and *vice versâ*. The part of the curve from *c* to *d* shows that the heart contracts more slowly at that part than it did at the beginning of systole. As

we shall see presently this part of the contraction curve corresponds in time to a very high intra-ventricular pressure, whereby greater resistance is offered to the shortening of the fibres of the heart-wall. The top of the curve from *d* to *e* shows that the ventricular wall remains contracted for a certain time after the active shortening has ceased. The duration of this period varies according to the rate at which the heart expels its contents: this rate, as we shall presently show, may vary greatly under different conditions. The expansion of the ventricle wall takes place with varying rapidity, the line from *e* to *f* descending at a fairly even rate. Sometimes the point *f* constitutes the lowest part of the curve (as in A and B of Fig. 2), while in other cases, (as in C of Fig. 2) the line of descent shows a marked shoulder, *g*, the part from *g* to *a* corresponding in time with the inflow of blood resulting from the venous and auricular systole. This shoulder is best-marked under conditions in which the venous pressure is low, and when, therefore, the amount of blood which can enter the ventricle before the auricular contraction is relatively small in amount.

Curve of Contraction taken in a line running round the Ventricle.

In Fig. 3 we give a myographic tracing from the wall of the left ventricle along a line parallel with the auriculo-ventricular

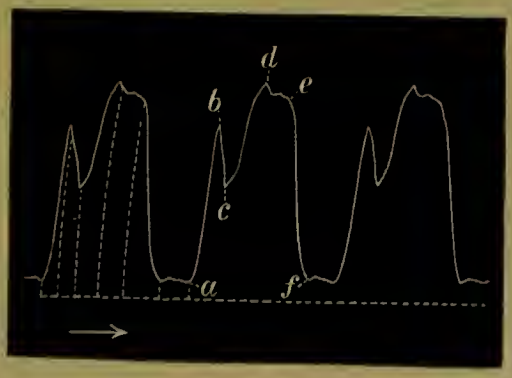

FIG. 3.

sulcus, taken, therefore, in a transverse direction, about half-way between the base and apex of the ventricle.

The tracing does not call for much remark. It resembles generally the myographic curve of the longitudinal fibres shown in Fig. 2. The main difference consists in the greater depth of the notch at *c*, and this, according to our experience, is an invariable characteristic of the transverse ventricular curve, as compared with the longitudinal curve.

Whatever be the cause of this notch, it acts more powerfully on the circular than on the longitudinal fibres.

SECTION II.

CONTRACTION CURVE OF THE PAPILLARY MUSCLES AND COMPARISON OF THIS WITH THE MYOGRAPHIC CURVE FROM THE WALL OF THE VENTRICLE.

To obtain trustworthy records of the contractions of the *musculi papillares* in the living mammalian heart is not such a hopelessly difficult matter as it may appear at first sight. We employed for this purpose a modification of the myocardiograph, by means of which it is possible to obtain curves of the contractions of the *musculi papillares* simultaneously with tracings of the contractions of the ventricular wall, the heart continuing, through the whole course of the experiment, to furnish these two curves without any appreciable abnormality in its action.

Fig. 1 represents the instrument arranged for this purpose. The end (*c*) of the light vertical rod (*a*) of the instrument is first of all tied to a point on the surface of the ventricle situated not far from the apex, and, as nearly as can be guessed, over the origin of one of the papillary muscles. The wire (*r*) is introduced into the ventricle through the auricular wall and the small hook at one of its ends is hooked over the free edge of one of the mitral flaps. It is perfectly easy to do this, although it may be necessary to move the hook once or twice in order to get it on to the middle of the flap chosen. The characters of the curve show when this has been effected. If it be hooked over the flap at a point where the latter is very narrow, the curve

obtained resembles that of the heart-wall. The middle part of the edge of the flap, on the other hand, is little affected by the contractions of the ventricle-wall, its movements being due almost entirely to the contractions and expansions of the papillary muscles.

The wire hook passes through the wall of the auricle, and, in order that there may be neither escape of blood by the side of the wire, nor interference with its movements by the auricular contractions, it is provided with a collar (*k*), in which it can slide easily, but which fits sufficiently closely to prevent escape of blood between the wire and the collar. The collar pierces the auricular wall, to which it is firmly tied so that no blood can escape. The hook can be inserted and kept in position throughout the experiment without a drop of blood escaping from the auricle. From the extra cardiac end of the wire a thread passes round a light grooved pulley (*m*), and then is carried upwards through the hole (*b*), at the centre of rotation of the rod (*a*), to the recording lever (*n*), being kept taut by a fine indiarubber thread (*o*), by means of which the degree of pull on the *musculi papillares* can be regulated at will. The longitudinal ventricular curve is obtained by means of the same instrument in the way already described.

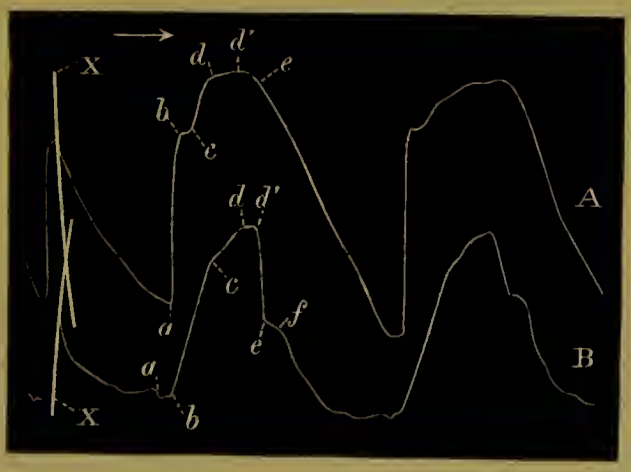

Fig. 4.

The curves in Figs. 4 and 5 show simultaneous tracings from the ventricle wall A, and the papillary muscles B, obtained in the

manner just described from the left ventricle of a small terrier dog. The lines X, X, give the position of the lever points when the drum is at rest, and points equidistant from them on the two curves correspond in time, so that, by means of a pair of compasses, it is possible to find with exactitude the relation in time of the two curves. These traces show that the contraction of the papillary muscles begins after that of the heart-wall. That part of the contraction of the ventricle wall which lies between *a* and *b* in curve A (Figs. 4 and 5) takes place before

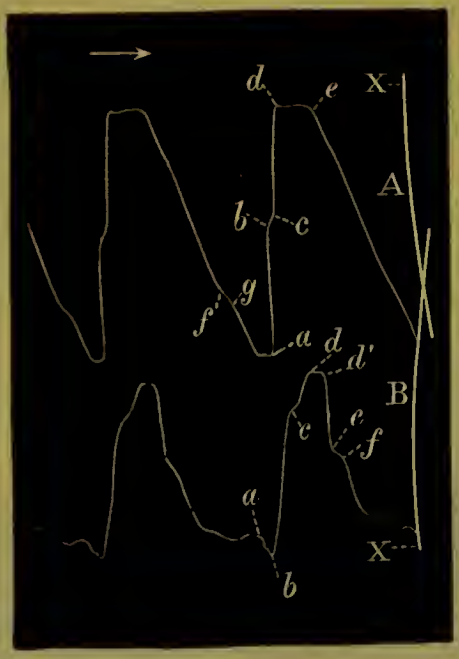

Fig. 5.

the contraction of the *musculi papillares* begins. The point *b* of the heart-wall curve corresponds exactly in time with the sudden commencement of the contraction of the papillary muscles. During the first part of the systole, then, the contraction of the longitudinal fibres of the ventricular wall approximate base and apex before the free edges of the auriculo-ventricular valves are pulled towards the apex by the contraction of the *musculi papillares*. This will of course result in a bulging upwards towards the auricle of the auriculo-

ventricular flaps during the period immediately following their closure. The edges of the valves may even be pushed away from the point of origin of the papillary muscles by the rise of intra-ventricular pressure during this first part of the systole, as is shown in curve B of Fig. 5. This must be due to passive stretching of the papillary muscles and of the *chordæ tendineæ*.

The shortening of the papillary muscles takes place in two successive stages. During the first of these (from *b* to *c* of Figs. 4 and 5) the contraction is rapid. This part of the curve corresponds in time to the arrest or slowing of the contraction of the ventricle-wall (*b* to *c* of the curves A) which is so constant a characteristic of these myocardiographic tracings.

To continue our analysis of these two sets of curves in Figs. 4 and 5—it can be seen that during the second part of the contraction of the papillary muscles (*c* to *d*) the shortening is slower than during the first stage. There are several very obvious reasons why this should be so. (1) When the edges of the mitral flaps are pulled down so that they are more or less in a line with the the *chordæ tendineæ*, the resistance to the contraction of the papillary muscles will be greater than when the valves are bulged upwards towards the auricle. (2) If the continued contraction of the ventricular walls keep the intra-ventricular pressure fairly high, after the papillary muscles have nearly reached their maximum shortening, the papillary muscle is placed at a disadvantage, and may even undergo passive stretching, resulting in the appearance of a notch between *c* and *d* of the papillary curve. In other words the papillary muscle, during the first stage of its contraction, is so well placed in relation to the ventricle wall that its contraction usually causes passive arrest or even retrogression of the shortening of the fibres of the latter; while during the second phase of contraction of the *musculi papillares* (from *c* to *d* of Figs. 4 and 5) they are on a more equal footing with the ventricle wall. It need hardly be said that the degree of distension of the ventricle at this part of the systole will influence greatly the degree to which one of these two competing elements of the ventricular systole will be effective at the expense of the other.

The third phase of the contraction of the *musculi papillares* is that of persistent contraction (*d* to *d'*) unaccompanied by continued shortening. This causes the curve to be more or less flattened at the top.

At the point *d'* the relaxation of the papillary muscles begins, and this commencement of their relaxation precedes the beginning of the relaxation of the ventricular wall, as can be seen by a reference to Fig. 4, the letters on the two curves of which mark points which correspond in time. The contraction of the papillary muscle, therefore, not only begins later than that of the ventricular wall, but it comes to an end sooner. The period of contraction of the papillary muscles (from *b* to *d*) may be about half of that of the ventricle wall, although the difference in duration of the two is usually less than this, the relation being about 5 to 8; the relation at all events is not a constant one. This short duration of the papillary contraction as compared with that of the ventricle wall *might* be explained by a wave of contraction running through the muscle-fibres of the ventricle wall till it reached the papillary muscles, returning as a reflected wave. The facts at our disposal, however, do not permit of our proving or disproving this theory.

It can be seen (in Figs. 4 and 5) that the expansion of the papillary muscles is at first rapid, namely, from the point *d* to *e*. During this period the ventricular wall still continues contracted. After *e*, the expansion of the papillary muscle is more gradual. Immediately after the point *e* on the papillary muscle curve B there is a more or less rounded shoulder *f*, indicating that at the commencement of relaxation of the heart-wall there is a slowing of the expansion of the papillary muscle. As the sudden fall of pressure within the heart begins with the commencement of expansion of the heart-wall, and not, to any appreciable extent, with the beginning of expansion of the papillary muscles, it is easy to understand why, while the intraventricular pressure is high, the papillary muscles expand more rapidly than when (after the point *e*) the intra-ventricular pressure is very much less. The shoulder *f* is presumably due to the sudden cessation of the pull exerted upon the *musculi papillares* by the pressure of the blood against the mitral valves.

After the point *f* the expansion becomes more and more gradual, ceasing before the expansion of the heart-wall has reached its maximum.

The analysis of the curves which we have given above enables us to divide the ventricular *systole* into five more or less distinct phases.

I.—During this phase the ventricle wall is contracting, but the *musculi papillares* are at rest (*a* to *b*, Figs. 2, 3, 4).

II.—During this the papillary muscles carry out the first rapid part of their contraction, accompanied by slowing, arrest, or retrogression of the shortening of the fibres of the heart-wall, which is most marked in tracings taken along a line running around the heart transversely (*b* to *c*, Figs. 2, 3, 4, 5).

III.—During this the shortening of the papillary muscle is considerably less rapid than during the last phase; the fibres of the heart-wall are also being shortened, although much more slowly than during the first part of its systole (*c* to *d*).

IV.—During this phase both papillary muscle and ventricle-wall remain contracted, but do not undergo further shortening (*d* to *d'*).

V.—During this period the papillary muscle expands rapidly, while the ventricle-wall remains contracted (*d* to *e*).

This last phase belongs both to systole and diastole, the expansion beginning in the papillary muscles.

The *diastole* of the heart-wall may be divided into three phases, namely:—

I.—During this expansion takes place rapidly, and with fairly uniform rapidity (*e* to *f*).

II.—This phase is only well shown on the curves when the amount of blood available to permit the ventricle to expand is not great, so that the expansion becomes slowed or even arrested after the first elastic expansion of the ventricles has drawn into them the greater part of the available blood (*f* to *g*, Fig. 2, C and 5 A).

III.—During this phase the wave of blood which results from the contraction of the veins and auricles reaches the ven-

tricular cavity, and causes or allows the final expansion of the ventricular wall which precedes systole (*g* to *a*).

It will be observed that we have said little or nothing as yet as to the absolute or relative duration of these eight phases of the ventricular cycle. The reason of this is that the relative duration is not by any means a fixed one, and the subject can therefore be best considered when we come to speak of the influences which vary the character of the heart-beat.

HEART-BEAT AND PULSE-WAVE.

BY C. S. ROY, M.D. F.R.S.,

Professor of Pathology,

AND J. G. ADAMI, M.A., M.B.,

Demonstrator of Pathology, in the University of Cambridge.

[From the *Cambridge Pathological Laboratory.*]

SECTION III.

THE CURVE OF INTRAVENTRICULAR PRESSURE, AND COMPARISON OF THIS WITH THE MYOCARDIOGRAPHIC CURVE.

THESE curves can only be satisfactorily compared when they are taken simultaneously from the same ventricle. To obtain the curve of intraventricular pressure we made use of the instrument employed by Rolleston, which gives, we have reason to believe, correct tracings of the changes of pressure within any cavity of the heart with which it is placed in communication.

This instrument, which is represented in Fig. 6, has been described by Rolleston.[1] We need only say here that the light

[1] *Journal of Physiology*, vol. viii. (1887) p. 295.

piston, *b*, which can oscillate in the wide metal tube communicating with the cavity of the left ventricle by the canula (shown in the lower part of the figure), is connected above to the recording lever (shown broken). The instrument is filled with oil or salt solution, the escape of which by the side of the piston is prevented by the accurate adaptation of the piston within the tube. The lever is attached to the thin strip of steel, *a*, whose torsion corresponds with the pressure to which the piston is subjected. The canula of the instrument is introduced into the left ventricle, either through the apex or through the

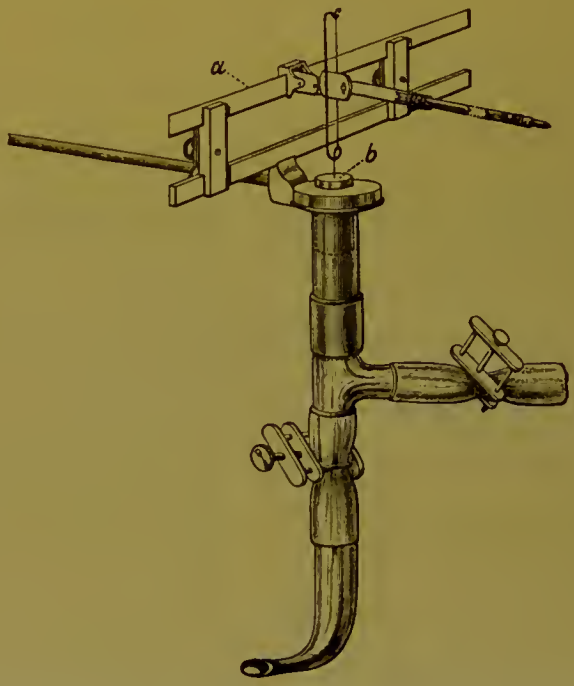

FIG. 6.

auricle. It might be connected with the ventricle by means of a tube passed down the carotid, but the friction of the fluid in a narrow tube of the required length would introduce a serious source of error.

Rolleston proved that pressure-curves from the ventricle are the same in character whether the thorax be opened or unopened. Pressure-curves from the left ventricle are shown in Fig. 7 and Figs. 9, 10, 11, and 12 B. Although they have already been described, we think it is desirable to say something here of their characteristics. The rise of pressure begins at *a*, the ascent

being sometimes at first somewhat slow, until the point *a* (Fig. 7) or *a'* (Fig. 9) is reached, after which the rise is rapid to *c*, which is usually the point of maximum pressure. The first slow rise from *a* to *a* is not due to the auricular contraction, this being sufficiently proved by the fact that the point *a* corresponds in

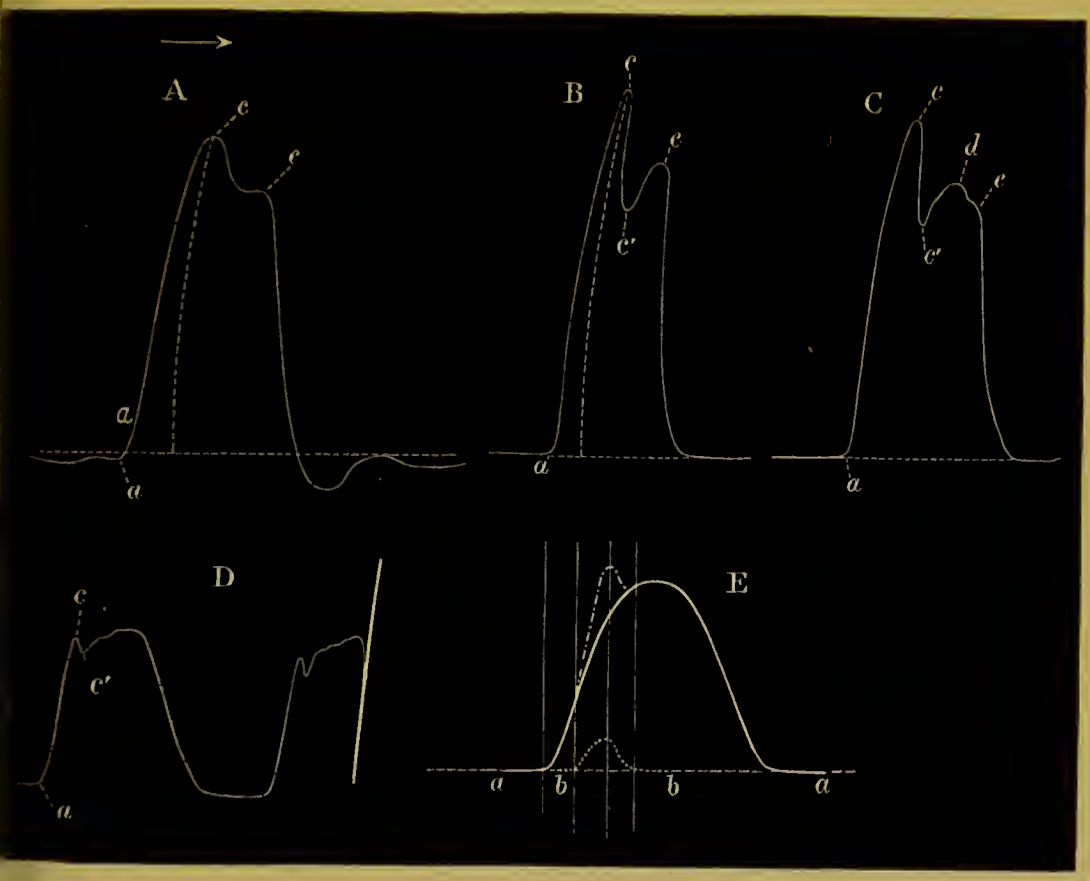

FIG. 7.

A, curve of intraventricular pressure, left ventricle of dog; B, C, D, similar curves obtained after exhibition of medicinal doses of tincture of strophanthus; E, diagrammatic representation of component parts of the systolic rise of intraventricular pressure.

time with the commencement of contraction of the ventricular wall, as can be seen in Fig. 9. From the absence of any wave coincident in time with the auricular systole (save in very exceptional cases, as in Fig. 7 A), it would seem evident that the resistance offered by the ventricles to the inflow of blood from

the auricles is in general so slight that no appreciable rise of pressure results from the auricular contraction.

This absence of any appreciable rise appears at first sight a contradiction to what is already known regarding the function of the auricles. It does not, however, imply that no increase in the inflow of blood into the ventricle takes place at the moment of auricular systole. As will be seen presently, the contraction of the veins and auricles causes a by no means slight acceleration of the flow of blood through the auriculo-ventricular orifices. What these curves do show is, that the auricular contraction causes little or no increase in the intraventricular pressure. As the curves first published by Chauveau and Marey, representing the intracardiac pressure in the horse's heart, are those upon which the most usually received views upon the intraventricular pressure are based, it may be as well for us to point out that the curves in question are in some respects apt to mislead.

The method employed by them, employing as it did a recording "tambour," connected with a thin-walled indiarubber bladder in the ventricle, the apparatus being filled with air, necessarily involves an exaggeration of slight changes of pressure when the pressure is at, or but little above, that of the atmosphere. Consequently the part of their intraventricular curve which corresponds in time to the auricular contraction is enormously exaggerated, while the upper part of the curve, corresponding to the condition of full contraction, inadequately represents changes of pressure. This can be very well seen in Fig. 8, which we reproduce from Marey.[1] This curve gives a record of the intraventricular pressure from a horse, and at the same time shows the true value of the different heights of the tracing. It is evident that the auricular systole does not raise the pressure in the ventricle by as much as 10 mm. of mercury. When in the case of a large heart like that of the horse the auricular systole affects the ventricular pressure so very slightly, it is not be wondered at that the rise of pressure in the smaller heart of the dog or of man is insufficient to affect the curve obtained with the more correct pressure-gauge employed by Rolleston and ourselves.

This absence of rise of pressure is due partly to the fact that

[1] *Circulation du Sang* (1881), p. 111.

there are no valves at the openings of the large veins into the auricles, such as would allow of the auricular pressure raising that within the ventricle to any great extent; and partly to the readiness with which the ventricular walls expand and accommodate themselves when more blood enters during the period of diastole. As can be seen from the tracings C and D, Fig. 7, the commencement of the systolic rise of pressure is sometimes abrupt. The first slow rise, upon which we have already dwelt to some extent, may, we imagine, be safely assumed to be due (when present) to the elastic stretching of the *musculi papillares;* these, as we have mentioned in Section II., are passively elongated previous to their contraction It

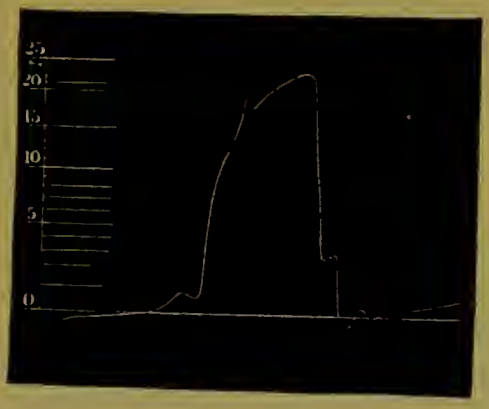

FIG. 8.

Intraventricular pressure-curve from left ventricle of horse (Marey)

will be remembered that we have shown that the contraction of the papillary muscle commences after that of the ventricular wall.

After the maximum pressure is reached at *c*, the pressure falls to a varying extent to the point *c'*, forming afterwards a more or less rounded shoulder between *c'* and *e*.

The height of the superposed wave *c* above the shoulder (*c'* to *e*) may vary greatly under different conditions, the wave may even appear on the ascending limb of the curve, as in tracing D, Fig. 7; or, as can be seen in B and C, Fig. 7, taken after the administration of a medicinal dose of strophanthus, this wave may rise high above the summit of the rest of the pressure

curve. That this superposed wave is not due to any inertia-vibration of the recording apparatus is sufficiently shown by the character of the tracings. We have, moreover, made control experiments with the recording instrument, which show that the moving parts of it are prevented from showing inertia-vibrations, owing to the friction between the piston and the walls of the tube in which it oscillates.

The intraventricular pressure-curve is, then, made up of two waves: one whose apex is at *c*, of short duration, and another and flatter one, upon which the former is superposed. In other words, the curve is composite, being formed of two waves that do not bear a constant relation to one another. The diagram E, Fig. 7, may serve to illustrate our meaning. The lines *aa* and *bb* show two pressure-curves as they would appear dissociated from one another; while if these be combined they form an interference curve. As two such waves appear upon the *sphygmographic* tracing, it is desirable to know exactly how they are produced. The relationship in time of the various parts of the ventricular pressure-curve to those obtained from the heart-wall and papillary muscles indicates clearly enough the mode of their production.

Fig. 9 gives a simultaneous tracing of intraventricular pressure and myocardiographic curves, the latter taken in a line from base to apex. It can be seen that the wave *b*, *c*, *c'*, in curve B, corresponds in time to the interruption between *b* and *c'* of the ventricular wall curve. [The letters upon the two curves represent points which correspond in time.] This wave, then, cannot be due to the contraction of the muscular wall of the ventricle, seeing that it appears at a time when the shortening of the fibres of the ventricular wall is interrupted.

As already mentioned in our last Section, the interruption, *bc*, of the myocardiographic curve coincides in time with the rapid shortening with which the papillary contraction commences. As this is the only part of the ventricular muscle which is undergoing rapid contraction at this phase of the systole, it is obvious that the superposed pressure-wave *c* is due to the pulling down of the flaps of the auriculo-ventricular valves, which, as we have shown, are bulged upwards during the first part of systole And the arrest of the contraction of the heart-wall (*b* to *c* in A)

is obviously due to the increased resistance to the shortening of the fibres of the ventricular muscle caused by the rise of pressure which results from the sudden pulling down of the mitral flaps.

The explanation of the fall of pressure from *c* to *c'* will be

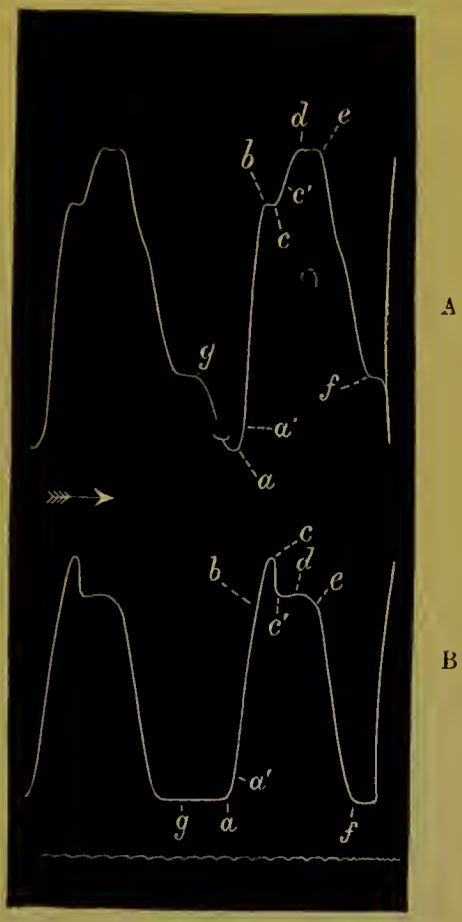

FIG. 9.

A, myocardiographic tracing from wall of left ventricle (dog); B, intraventricular pressure curve from left ventricle obtained simultaneously.

most conveniently considered when we come to refer to the pulse-wave. Suffice it to say here that it is due to the escape of blood into the aorta. The escape of blood into the aorta before the point of maximum pressure is reached explains the slowing of the rise of pressure which can be seen in the upper part of the ascending line in Fig. 8, and to a less extent, though still appreciably, in C, Fig. 7.

The shoulder *c'* to *e* in B (Fig. 9) coincides in time with the part of the heart-wall curve (A) lying between the same letters, and during its first portion the ventricle wall continues to contract, as do also the papillary muscles, although the shortening of both is slow. For a varying time at the end of this period the shortening of the heart-muscle has ceased, and of course, therefore, the outflow from the ventricle. The point *e* at which the pressure falls corresponds with the point *e* on the muscle-curve, at which expansion begins. As we have pointed out, the expansion of the papillary muscles precedes that of the heart-wall, and this is presumably the reason why the pressure curve is more rounded at *e* than could be expected from the suddenness of the expansion of the heart-wall. Under some conditions, as indicated in curve C, Fig. 7, there is a notch between *d* and *e*, separating the fall due to papillary expansion from that due to the expansion of the heart-wall.

In the first three curves of Fig. 7 the horizontal dotted lines represent the atmospheric pressure. It can be seen that in only one of them is there any marked fall of the intracardiac pressure below that of the atmosphere—a fall corresponding in time with the interruption of the diastolic expansion of the ventricle (*f* to *g*) in certain heart-wall curves (for example, in Fig. 9, and in Figs. 2 C, and 5 A, in Sections I. and II.).

The four curves A, B, C, D, in Fig. 7, are taken from different animals. As there is no reason for supposing that curves B, C, and D are less trustworthy records than curve A, it follows that a phase of negative pressure during diastole is not of constant occurrence; that it is due, in other words, to conditions of the heart and circulation not necessarily present within physiological limits. At what phase of diastole, first of all, does such negative pressure show itself? It is generally supposed that it coincides with the commencement of diastole, mainly, we imagine, because the elastic resilience of the ventricular wall on the cessation of systole has naturally been assumed to act most powerfully at that time—an assumption which appears to us quite reasonable. There is however another factor which plays of necessity an important part in leading to the negative pressure in question; we refer, namely, to the blood which is available for filling the ventricle. If the large veins be dis-

tended with blood, ready to flow into the ventricle *pari passu* with its expansion—if for example there is a fairly high venous pressure—it is not very easy to see how there can be produced any considerable negative pressure, and still more how a negative wave which lasts so long as that in curve A can be produced, if there be blood available for filling the vacancy in the ventricular cavity. During the ventricular systole the blood does not cease to enter the auricles from the large veins, and the amount of blood thus stored is available for filling the ventricular cavity during the first part of the diastole. As can be seen in curve A, Fig. 9, the interruption of the expansion (*f* to *g*), which, as already mentioned, corresponds in time with the phase of negative pressure inside the heart-cavity, occurs at a period when the heart-wall has already undergone very considerable expansion—in this curve after about three-fourths of the total diastolic elongation of the ventricular muscle fibre. The occurrence of negative pressure at this period and the resulting interruption of the diastolic expansion can only be due, so far as we can see, to there not being a sufficiently large blood-supply to keep pace with the elastic dilatation of the heart.

This explanation of the negative pressure implies that it occurs at a phase of diastole during which the flow of blood through the auriculo-ventricular orifices (if indeed it be not arrested) is slower than during the first phase of diastole, when, although the suction power is presumably greater, it is unable to produce negative pressure owing to the inflow of blood which has collected in the auricle during the ventricular systole. During the latter part of the diastole there is of course an increased inflow of blood, owing to the successive contractions of the veins and auricles.

We may note in passing that the inflow of blood from the auricles at the moment of opening of the auriculo-ventricular valves may cause a positive wave on the line of descent of pressure (as can be seen in Figs. 8 and 11 B). In Fig. 8, where perhaps it is best shown, it occurs at a time when the pressure has fallen to about 30 mm. of mercury. In other cases, again, the inflow leads to a slowing of the fall of pressure (C, Fig. 7). In any case this inflow precedes the wave of sub-atmospheric pressure where this wave shows itself.

If therefore we consider the flow through the auriculo-ventricular orifice, we may divide it into four periods: the *first*, during ventricular systole, in which it is quite arrested, and in which, therefore, the auricles act as reservoirs; the *second*, that of rapid flow through the orifice resulting from the resilience of the ventricle (for any rise of pressure in the auricles, which may take place as a result of the accumulation of blood during the previous period, must be so slight that it may safely be ignored); the *third* stage is that during which, whatever the amount of blood available, there must be a slowing owing to diminished resilience of the heart-wall, the resilience diminishing with the ventricular expansion (if the amount of blood available be below a certain limit, the resilience of the wall of the ventricle may still suffice to produce a negative pressure, as above explained); the *fourth* phase is one of increased rapidity of flow through the orifice, owing to the successive contraction of the veins and auricle.

Let us consider for a moment how our conclusions upon this subject bear upon the murmurs which, when present, are characteristic of mitral stenosis. In this condition a continuous murmur extending from the second to the succeeding first sound is of infrequent occurrence, whereas a murmur following the second sound, or a presystolic murmur, that is, one preceding the first sound, is common enough; or there may be a combination of both, a pause intervening. With regard, first of all, to the murmur following the second sound. This occurs at a time when through the narrowed orifice there is a rapid inflow into the ventricles as a result of the resilience of its walls at the beginning of diastole. The velocity of inflow, which is the important factor in the causation of this murmur, will not be diminished to any extent as the result of a limited amount of blood being available to enter the ventricle, seeing that in mitral stenosis there is congestion of the pulmonary veins, and the supply of blood is correspondingly large; but it must diminish with the diminution in suction power of the ventricle which necessarily accompanies its expansion. This diminution in suction explains the pause which in certain cases intervenes between the murmur following the second sound and that which precedes the first. Whatever conditions, moreover, lead to dilatation of the left

ventricle, whether these consist in an increase of the work thrown upon it, as the result of aortic disease, or otherwise, or whether they are of the nature of a diminution of the power of contraction of the heart-wall, will necessarily result in a lessening of the suction force, and will therefore be less favourable for the production of this murmur. It is not, therefore, difficult to understand why it is that this murmur may appear and disappear in a given case, without there being any reason to believe that the condition of the orifice has undergone change, and also why, in many cases, it is absent throughout the whole course of disease. The presystolic murmur is of course due to increased rapidity of flow through the constricted orifice, resulting from the contraction of the pulmonary veins and left auricle, and when present it indicates that the force of auricular contraction is great enough to raise the pressure on the auricular side of the orifice to an extent sufficient to increase the rapidity of flow through the narrowed opening. The great variation which, as we shall show elsewhere, may take place in the contraction of the auricles, is quite sufficient to explain the appearance and disappearance of this murmur, without there being any accompanying change in the size of the orifice. Where the murmur lasts throughout the whole period of ventricular diastole, it must be due to a considerable narrowing of the orifice and the resulting great pressure in the pulmonary veins.

SECTION IV.

THE APEX-BEAT OF THE HEART

The cardiographic curves from man have not as yet been found fitted to give much information regarding the action of the heart either in health or in disease. That this is so is doubtless partly owing to the imperfect methods by which the curves have been obtained. Even when taken by so good a cardiograph as that of Von Basch, it is by no means easy at first sight to interpret the tracings, and to say what is the exact cause of each rise and fall. But over and above the instrumental difficulties, apex-beat curves are incapable of giving

more than a limited amount of information; we refer in the present connexion to curves taken from the area of cardiac impulse in man. Variations in the size of the organ will influence the extent to which the ventricle is in apposition to the thoracic wall, will also to some extent affect the force of the apex-beat, and will cause disproportionately great variations in the character of the tracings. The degree also to which the heart is wedged in between the diaphragm and the thoracic wall must introduce modification; moreover, if the most anterior portion of the ventricle lies behind a rib, the beat, as communicated to the recording instrument, will necessarily be less powerful than in the cases where the full strength of the beat is communicated through an intercostal space. Besides all this, differences in the degree of resistance of the intercostal tissues and of their thickness must, it need hardly be said, limit the advantages of a comparison of the cardiographic curves obtained from different individuals. Nevertheless, certain data which may be of considerable value to the physician can be obtained by the employment of an appropriate form of cardiograph, and it is therefore desirable that we should analyse this form of heart-curve with some care.

As we shall presently seek to show, such an analysis is greatly aided by taking simultaneous graphic records of the changes in the antero-posterior diameter of the ventricle and of the intracardiac pressure, or of the contractions of the heart-wall, employing for this purpose the dog or other animal in which it can be demonstrated that the heart-action and cardiac impulse correspond to that of man.

The necessary operation to gain this end may here be briefly indicated. Having made a window in the chest-wall, with all the necessary precautions already referred to, the pericardium is divided along the anterior aspect of the heart, and its edges are firmly secured by stitches to the sides of the window, so that the ventricles rest by their posterior aspect upon a secure and immobile floor formed by the hinder portion of the pericardial sac, this having been rendered somewhat tense by the process of stitching the divided anterior portion to the chest-wall. The ventricles now may be looked upon as fixed posteriorly, and if, to the anterior surface of the left ventricle, there be attached the

button-like termination of a light lever, the up-and-down movements of the lever can by very simple means be recorded upon a revolving smoked drum, and in this manner there can be gained a tracing of the variations in thickness of the ventricles—that is to say, of the changes in the antero-posterior diameter of the heart. Further, by the action of a spring or elastic band, the lever above mentioned may be made to exert a pressure upon the ventricular wall, the extent of which can be varied at will. Such a pressure is necessary in order to differentiate between the passive increase in the antero-posterior diameter of the flaccid ventricle during diastole, and what may be termed the active increase in systole. We may say that in diastole the flaccid heart tends to accommodate its shape to that of the surrounding cavity; thus under normal conditions, when the chest-wall is intact, the pressure upon it anteriorly and posteriorly, of the chest-wall and diaphragm respectively, causes the transverse diameter to be the more considerable. In systole, on the other hand, with the contraction of the ventricles the walls become tense and resisting, the organ becomes rounded, the transverse diameter is diminished, the antero-posterior diameter increased. Hence pressure exerted on the front of the ventricles reproduces the conditions to which the heart is normally subjected, and the curve obtained resembles the cardiographic tracing in man.

While recording the changes in the breadth of the heart, a simultaneous record may be made either of the intracardiac pressure by Rolleston's instrument, in the manner previously described, or of the contraction of the ventricular muscle by the heart-wall apparatus; and the phases of the curves recorded simultaneously may in this way be studied and compared.

The first question to be considered is whether the cardiographic curve in man differs in any way from similar curves gained from lower animals. In Fig. 10 (1) is represented a typical trace from the human heart, copied from an illustration given by François Franck, obtained from a case of ectopia cordis. Curves similar in character have recently been obtained by Dr. Hercules MacDonnell, from the heart of a patient whose abdominal walls were so greatly relaxed after the evacuation of ascitic fluid that he was able to push up his

hand between liver and diaphragm, and apply a tambour and recording arrangement to the posterior aspect of the heart.[1] Side by side with the tracing from the case of ectopia cordis we give a tracing from the heart of a cat, which was obtained by resting a light recording lever on the anterior part of the wall of the left ventricle, the organ having been exposed by making a window in the thorax. A glance at these two curves will show that they are identical in character; in each a rounded

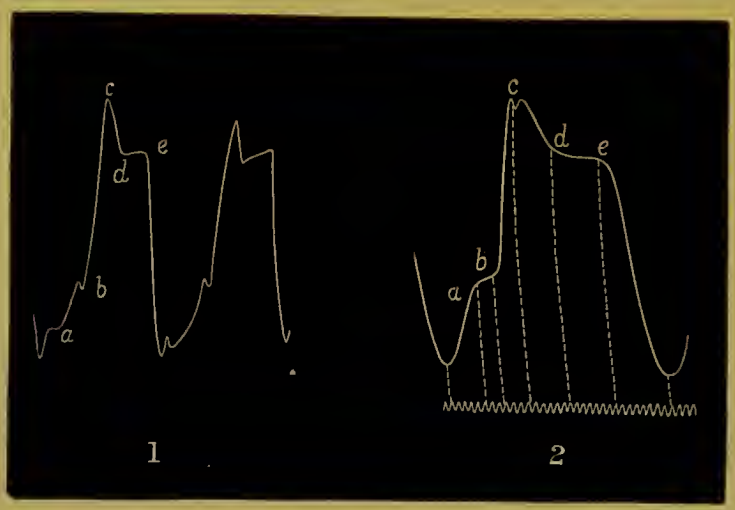

FIG. 10.

1, Cardiographic tracing from case of ectopia cordis (François Franck);
2, Cardiographic tracing from exposed heart of cat.

wave (*a* to *b*) is followed by a rapid ascent of the curve (*b* to *c*), while the summit shows a notch (*d*) and a more or less rounded shoulder (*e*) preceding the descent. In analysing the curves

[1] Some time ago Dr. MacDonnell placed before us the cardiographic curves which are reproduced in the present number of the *Practitioner* (p. 178). He requested us to make some remarks on the character of his curves, and on the conclusions which could be drawn from an analysis of them. On proceeding, however, to look over our own curves with this object in view we found so much regarding the mechanism of the heart-beat in mammals which seemed new, that we thought it better to go more deeply into the subject than would be possible within the limits of a note to be appended to Dr. MacDonnell's communication. As in the latter part of this Section we propose to go over the characters of the mammalian apex-beat curve, we need not here do more than congratulate Dr. MacDonnell on the success which has crowned his efforts to obtain trustworthy cardiographic curves from the human heart, by a method which has not hitherto been employed. It will be seen that his curves resemble those which we have reproduced in Fig. 10 from a case of ectopia cordis and from the heart of a cat.

obtained by recording the change in the antero-posterior diameter of the exposed mammalian heart, it may safely be assumed that our conclusions apply with equal effect to the apex-beat in man.

In Figs. 11 and 12 are given two simultaneous tracings of the

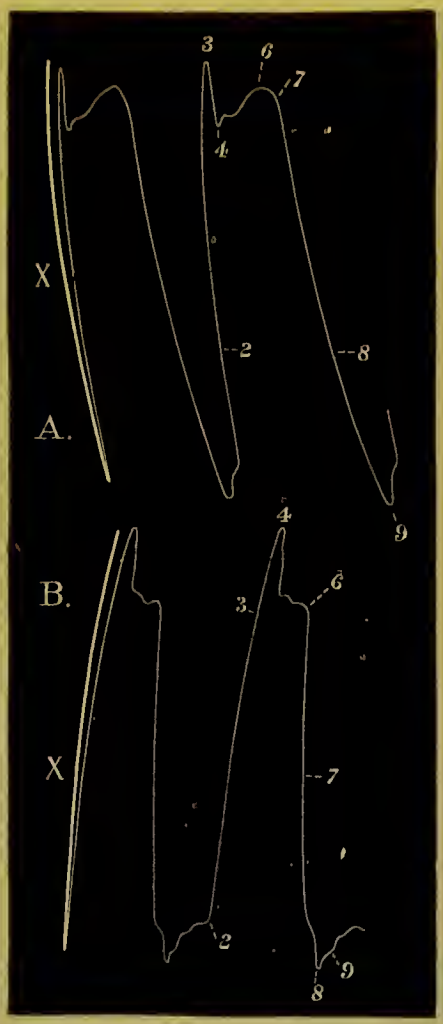

FIG. 11.

A, cardiographic curve of apex-beat; B, intraventricular pressure, taken simultaneously.

intraventricular pressure and the apex-beat (or more correctly of the changes in the antero-posterior diameter of the ventricles) taken from hearts of two dogs. In both of them the apex-beat, A, is sufficiently characteristic; they differ from one another and from the typical curve from the cat in Fig. 10 (2), in certain

particulars. The height of the curve, for example, is much greater in Fig. 11 than in Fig. 12. Such changes in height are due in a great measure to the degree of pressure which is exercised on the ventricular wall by the recording apparatus.

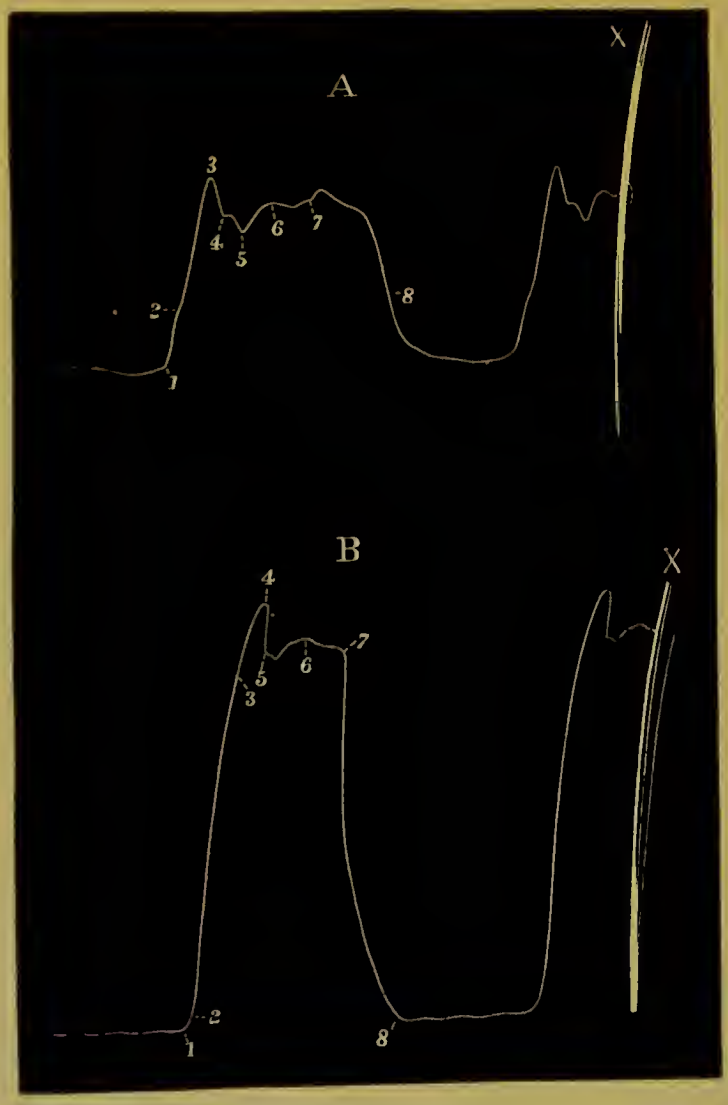

FIG. 12.

A, cardiographic curve of apex-beat; B, intraventricular pressure, taken simultaneously.

It is not the height of the curve however which alone is influenced by variations in the degree of pressure; the form and duration of the rise which accompanies the systole may also be very considerably influenced thereby, so much so that

measurements of the duration of the systole obtained from curves of the apex-beat in man must be comparatively worthless. Moreover the point of the ventricular wall upon which the recording arrangement rests exercises an extremely important influence upon the form of the tracing obtained. By varying the position and the pressure of the button of the recording arrangement an endless variety of curves may be obtained, which it would not be profitable for us to attempt to describe. When it is remembered how many and how complicated are the conditions which affect the apex-beat, or the antero-posterior diameter of the ventricles, it is very easy to understand why this form of graphic record of the heart's action varies so greatly.

HEART-BEAT AND PULSE-WAVE.

BY C. S. ROY M.D., F.R.S.,

Professor of Pathology,

AND J. G. ADAMI, M.A., M.B.,

Demonstrator of Pathology, in the University of Cambridge.

[From the *Cambridge Pathological Laboratory.*]

SECTION IV.—Continued.

We have said that it would not be profitable for us to seek to describe all the varieties of apex-beat curves, while to give any one of them as being a more true representation of the movements of the heart than the rest would only mislead the reader. We think it better, therefore, to begin by referring to a diagrammatic representation of the apex-beat curve, showing the changes in the character and height of the tracings which can be obtained with varying degrees of pressure exerted upon the heart-surface. It will, we believe, be found easy to interpret the main features of any individual tracing of the apex-beat if once this key (as we may call it) to such curves has been understood.

The diagram (Fig. 13,) shows three superposed tracings which represent the apex-beat curve, as recorded with three different degrees of pressure of the button on the surface of the heart.

With *slight* pressure, the lever-point during diastole occupies a higher position than when the pressure is greater, which is of course only what one would expect, the heart being less flattened. Moreover during the whole diastolic period the tracing slopes upwards, as the result of the inflow of blood from the auricles. With the auricular contraction the antero-

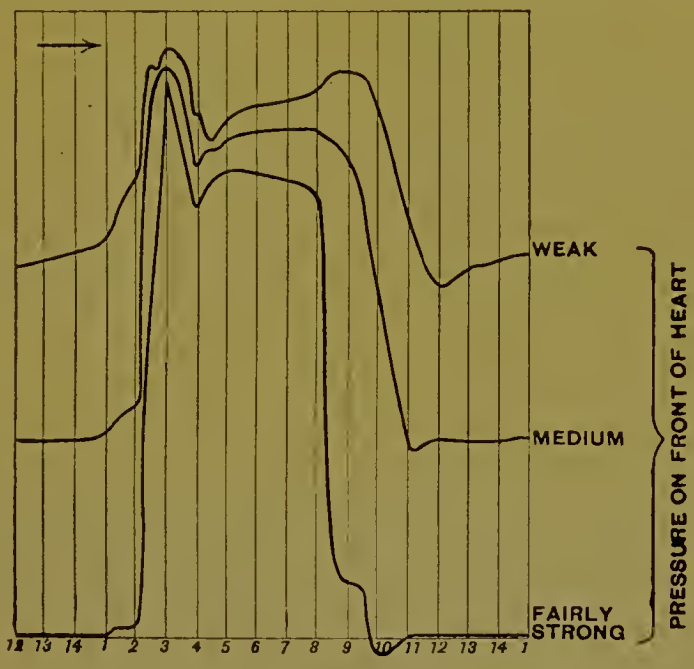

FIG. 13.

Diagram showing the effect on apex-beat curves of different degrees of pressure on the heart-wall.

posterior diameter of the ventricles is still further increased, causing a rapid but rounded rise (from 1 to 2). Immediately following the auricular wave there is an abrupt ascent due to the change in form of the organ resulting from its contraction. At the top of this ascent there is usually a notch (between 2 and 3), the small wave preceding which corresponds in time to the commencement of outflow from the heart. The relation in height of this little wave to the one following the small notch varies greatly according to the part of

the ventricle upon which the button rests, as well as upon the degree of pressure exercised by it, so that the relation of these two indicated in the diagram by no means applies to all such tracings. After the second of these waves the tracings always show a more or less abrupt descent (to 4). This point (4) may form the bottom of the main notch (*d*, in Fig. 10), as is usually the case when the pressure is greater; but on the whole, perhaps, the form shown in the diagram is more common where the pressure is very slight. In such cases, after this last small notch (at 4), the lever-point descends a little further, the descent being succeeded by a rise leading to a more or less flattened top (from 5 to 10). At the end of this a rounded shoulder precedes the fall of the lever-point, which results from the relaxation of the ventricular walls. This descent generally terminates in a notch (at 12).

If the pressure of the button be a *little stronger* the curve is modified in various particulars. During diastole the lever-point shows little, if any, rise, owing to the fact that the greater pressure on the part of the button prevents its elevation while the heart-wall is still flaccid. The auricular contraction also does not cause so great a rise of the lever: the line of ascent (from 2 to 3), on the other hand, is higher than when the pressure on the organ is slight. The summit of the curve, moreover, does not usually exhibit a notch between 2 and 3, although often enough the curve at this point has a rounded slope, evidently corresponding to the first wave, which is seen at the top of the curve obtained by slight pressure. From 3 to 4 there is a descent of the lever-point to a well-marked notch, this being followed by a sloping upstroke ending in a more or less level plateau. This passes by a less rounded shoulder than on the curve first described to the descent (9 to 10). The descent may be interrupted by a wave immediately after 10, after which the curve falls to the notch at 11.

With *still greater* pressure on the heart the apex-beat curve exaggerates the characteristics of this last described curve: no real rise of the lever-point occurs as a result of the inflow of blood from the auricle at the moment of its contraction, there being only a slight wave between 2 and

3, and the upstroke is longer. There is a notch at 4 followed by a more or less rounded plateau, passing by a sharp shoulder at 9 to the descent, which is not interrupted by a wave as in the curve taken with medium pressure, but which like the others terminates in a notch at the base; this, we may remark, is possibly due to inertia.

It will be observed that with low pressure the rise of the lever lasts a longer time than with stronger pressure. And the reason of this is obviously to be found in the fact that the distortion of the ventricles during diastole by the pressure of the recording apparatus will be overcome at an earlier period of the contraction of the heart-wall if the pressure on the wall be slight. The elevation of the lever will also last longer, until in fact the relaxation of the ventricle-walls at the end of systole advances sufficiently far to allow the walls to be flattened by the slight pressure of the button. With greater pressure there is, of course, greater indentation of the heart-wall during diastole, and as a consequence a greater rise of the lever during systole. The reason of this is, that the upper part of the curve, which corresponds to powerful contraction of the ventricular wall, and to a time when the pressure of the recording apparatus can distort the form of the heart very little, is nearly as high with fairly strong pressure as it is with weak. The result of this relationship between the distorting force of the recording apparatus (or in the case of the heart in man, of the pressure of the organ against the thoracic wall, or the wedging of it in between the latter and the diaphragm), and the force of contraction at different parts of the heart's cycle, necessarily is that, with slight pressure the upstroke of the lever begins earlier, and reaches its highest point earlier, than when the pressure is greater. The descent also begins later and ends later. Our diagram illustrates but does not exaggerate these variations in the commencement and termination of the ascent and descent of the tracing.

One thing to be kept in mind, then, with regard to apex-beat tracings is that in them *it is difficult, and, indeed, in most cases, impossible to measure with accuracy the duration of the different phases of the cardiac cycle.*

The top of the curves lying between 2 and 4, and especially

the notch at 4, the explanation of which is not at first sight very easy, now require to be considered. They can be most readily interpreted by comparing simultaneous tracings of the apex-beat (antero-posterior diameter) and of the intraventricular pressure. We give examples of such curves in Figs. 14 and 15.

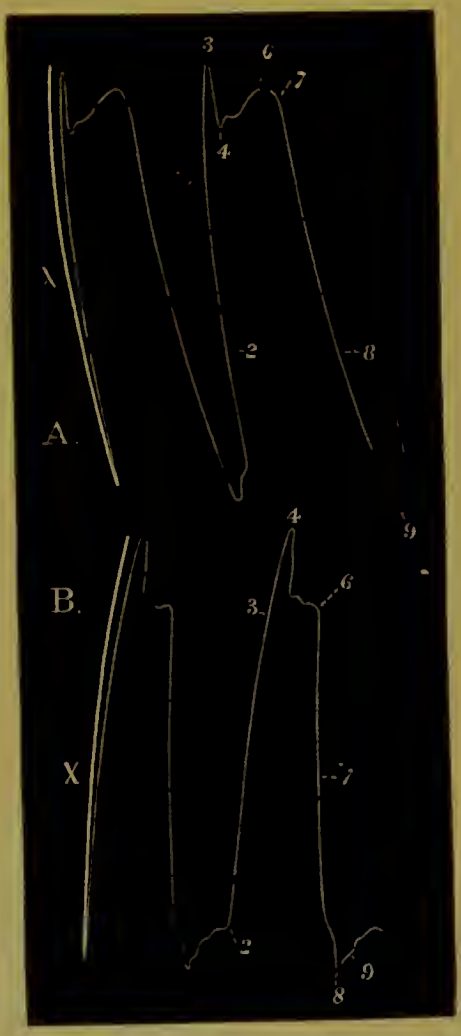

FIG. 14.

A, cardiographic curve of apex-beat, with fairly strong pressure on the heart-wall ; B, intraventricular pressure, taken simultaneously.

On measuring out the relations in time of these two curves it is found that the highest point of the apex-beat tracing (A of these Figs.) does not correspond with the maximum pressure within the ventricle, but that the latter occurs at the same

instant of time as the notch (4) of the apex-beat curve. This relationship appeared to us at first sight so very paradoxical that we measured out a large number of tracings, and made a few control experiments, in order to satisfy ourselves of its

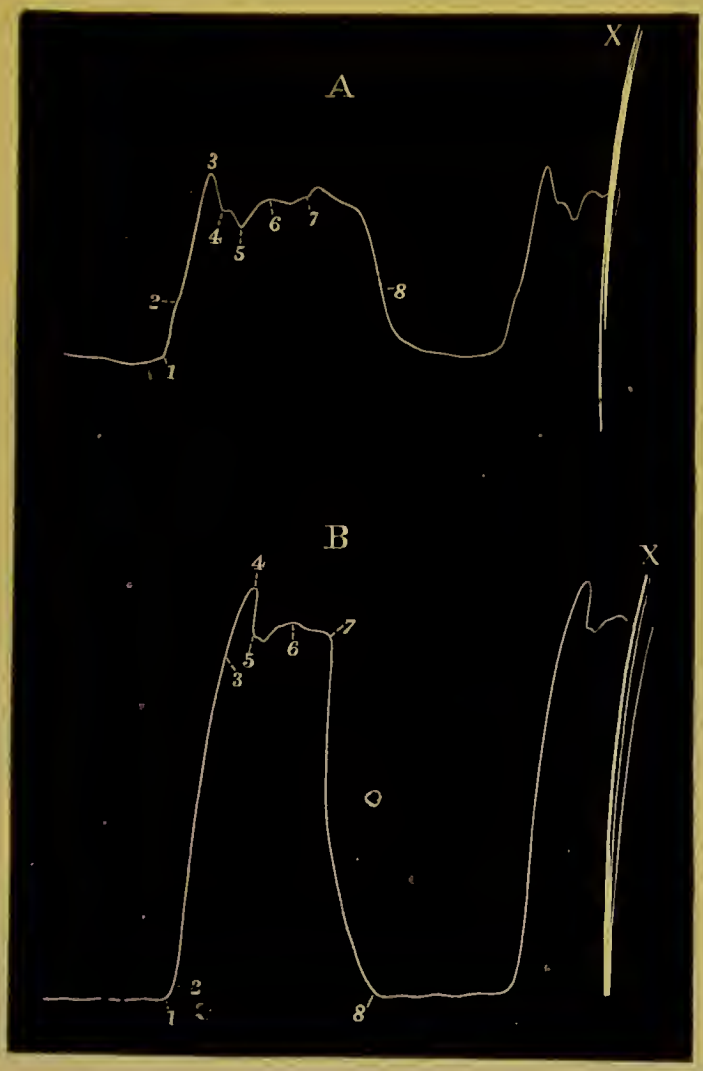

FIG. 15.

A, cardiographic curve of apex-beat, with slight pressure ;
B, intraventricular pressure, taken simultaneously.

accuracy. There can, however, be no doubt of the fact that the notch (4, in A, Figs. 14 and 15, and *d*, *d*, Fig. 16), which is so characteristic of cardiographic curves of the apex-beat, corresponds in time with the superposed wave 4 of B in

Figs. 14 and 15, and *c*, *c* in Fig. 16. If so, the question to be considered is, why does the first part of the apex-beat curve reach a higher level than the part at 4, which corresponds to a higher intraventricular pressure? The explanation is in reality simple enough. The antero-posterior diameter of the ventricles will reach its maximum as soon as the contraction of the ventricular wall has overcome the distorting forces which act upon it. As soon, however, as the blood begins to leave the ventricles by the aorta and pulmonary arteries, this diameter will necessarily diminish. This explains the descent of the cardiographic lever from 3 to 4 of A, in Figs. 14

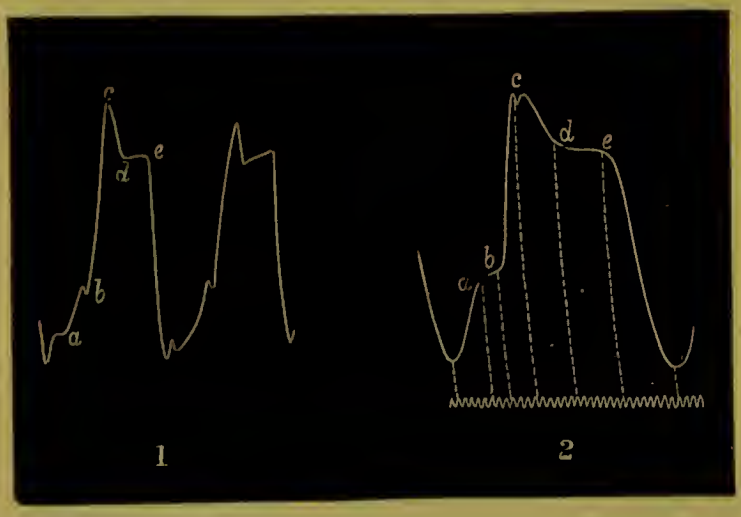

FIG. 16.

1, cardiographic tracing from case of ectopia cordis (François Franck); 2, cardiographic tracing from exposed heart of cat.

and 15, and *c* to *d* in Fig. 16. It will not, however, explain why there should be a well-marked notch at the moment of maximum intraventricular pressure. This notch is most marked in tracings obtained with the button resting near the apex of the heart, and it is, as need hardly be said, a characteristic feature of curves from the apex-beat of man. The notch corresponds in time with the rapid contraction of the papillary muscles, a contraction pulling down the auriculo-ventricular valves, the necessary result of which is that, although the intracardiac pressure is thereby raised, the part of the ventricular walls

from which the papillary muscles originate becomes indented.[1] With the further contraction of the heart-wall, continuing after the rapid contractions of the papillary muscles, these latter exert a lessened pull upon the heart-wall. This lessening of the pull, and the associated lessened distortion, is due to the diminution in sectional area of the auriculo-ventricular orifice, and also to the increased resistance to distortion on the part of the heart-wall which results from greater contraction. In this way is to be explained the rise of the lever-point which is often to be seen after the notch we have been discussing.

The part of the curve lying between 5 and 8 (Fig. 13) may or may not show waves and indentations. As these however, when present, vary with the point of the ventricle-wall on which the button rests, we have not indicated them in our diagram, and do not propose to say anything here regarding their cause. It will be seen that the curves taken with weak and with medium pressure of the button show a rounded rise after 8 in Fig. 13, 7 in Figs. 14 and 15, that is after the point at which, as can be seen from Fig. 15, the fall of pressure within the ventricle at the end of systole begins. The cause of this rise of the lever when the pressure is weak is evidently to be found in the fact that, during the earlier part of the diastolic expansion, the heart wall is not completely flaccid in other words that it still retains a certain amount of its power of resisting distortion, which goes hand in hand with

[1] We may note in passing that although the contractions of the *musculi papillares* do pull in the part of the ventricle wall from which the muscles originate, this distortion or indentation is not so great as would be the case were the ventricles globular instead of being of their characteristic conical shape. It must be remembered that a globular ventricle would be a more perfect pumping machine, inasmuch as a given degree of contraction of the wall would expel a larger quantity of blood than would be the case with any other form. That the vertebrate heart is conical rather than globular can evidently be explained by the fact that with a globular heart the papillary muscles would act much less efficiently. Were the heart spherical the contractions of the *musculi papillares* would be in great part expended in distorting the walls, and would therefore have less power to pull down the auriculo-ventricular valves, an act which, as we have pointed out, plays so very important a part in the expulsion of the blood from the ventricles. For the same reason that a Gothic arch can be more lightly constructed than a Norman one, in order to support a given weight, a conoidal heart is better fitted to allow the papillary muscles to act effectively, pulling as they do in the direction of the axis of the cone.

the degree of contraction of its walls. This not only prevents the lever falling when the pressure is slight, but results in a rise of the lever owing to the increase in the antero-posterior diameter of the heart resulting from the inflow of blood. It is this same resistance to distortion which acts as the suction-power, and in certain conditions produces negative pressure.

In the apex-beat curve obtained with fairly strong pressure (Fig. 13) we have shown a wave on the line of descent. This corresponds in time with the wave which is sometimes seen in the descending line of the intraventricular pressure curve—and both, like the waves referred to in the preceding paragraph, are due to the commencement of the inflow of blood from the auricles. This is shown in François Franck's figure (Fig. 16, 1) as a wave preceding the notch *a*.

From the above analysis of the apex-beat curve it will be gathered that the influences affecting it are very complex, but that it does give certain indications regarding the heart-beat. In the meantime what we have said above may at least help the physician to distinguish between modifications of the cardiogram which result from changes in pressure exerted on the heart by the thoracic wall and diaphragm, and those which are due to some deviation from the normal in the functional activity of the organ, of which latter we shall have something to say in a future Section.

SECTION V.

ON THE MECHANISM OF THE MAMMALIAN HEART-BEAT.

We are now in a position to put together the somewhat dry details which we have described in the preceding Sections. This can be perhaps most conveniently accomplished by superposing the various graphic records, of which so much has already been said, in such a way as to show at a glance the relations in time of the various factors which go to make up the complicated beat of the mammalian heart.

We have done this in Fig. 17, which shows (1) the contractions of the auricular wall, (2) the apex-beat curve, (3) the intraven-

tricular pressure-curve (4) the myocardiographic curve of the ventricle taken in a line joining base and apex, (5) a similar curve taken in a line parallel with the auriculo-ventricular valves, (6) the curve of contraction of the *musculi papillares*, (7) the periods of outflow of blood from and inflow to the ventricles (8) the positions of the first and second sounds of the heart, (9) the moment of opening and closing of the auriculo-ventricular valves, and (10) the times of opening and closure of the sigmoid valves.

This diagram has been constructed by measuring out simultaneous tracings in the manner indicated in the previous Sections.

It gives the relations in time of some of the chief elements that together constitute the beat of the heart, of which it shows one cycle. The manner in which most of the data on which it is based have been arrived at is sufficiently explained in the foregoing Sections. No explanation has however been given as to the manner in which we obtained information regarding certain other of the matters shown in the figure; for example, the exact positions in the cycle of the closure and opening of the auriculo-ventricular and sigmoid valves, the commencement and cessation of the inflow and outflow from the ventricles, and the positions of the first and second sounds of the heart.

In order that it may not be supposed that we have inserted the times of these events of the heart's cycle after merely thinking over the matter in the light of the facts related in the preceding Sections, it may be as well for us to give some indication of the method by which we have obtained information regarding them. The moment of opening of the sigmoid valves, and the commencement of the outflow from the ventricles, can be readily and accurately learned by measuring out simultaneous tracings of the intraventricular pressure and of the pulse-curve at the root of the aorta. Such curves also give exactly, as we will show later on, the time of cessation of the outflow. The time of closure of the auriculo-ventricular valves is of course that at which the pressure within the ventricles exceeds that in the auricles, and can therefore be easily obtained, while the moment of opening of these valves is often enough shown by a small wave present on the descending lines

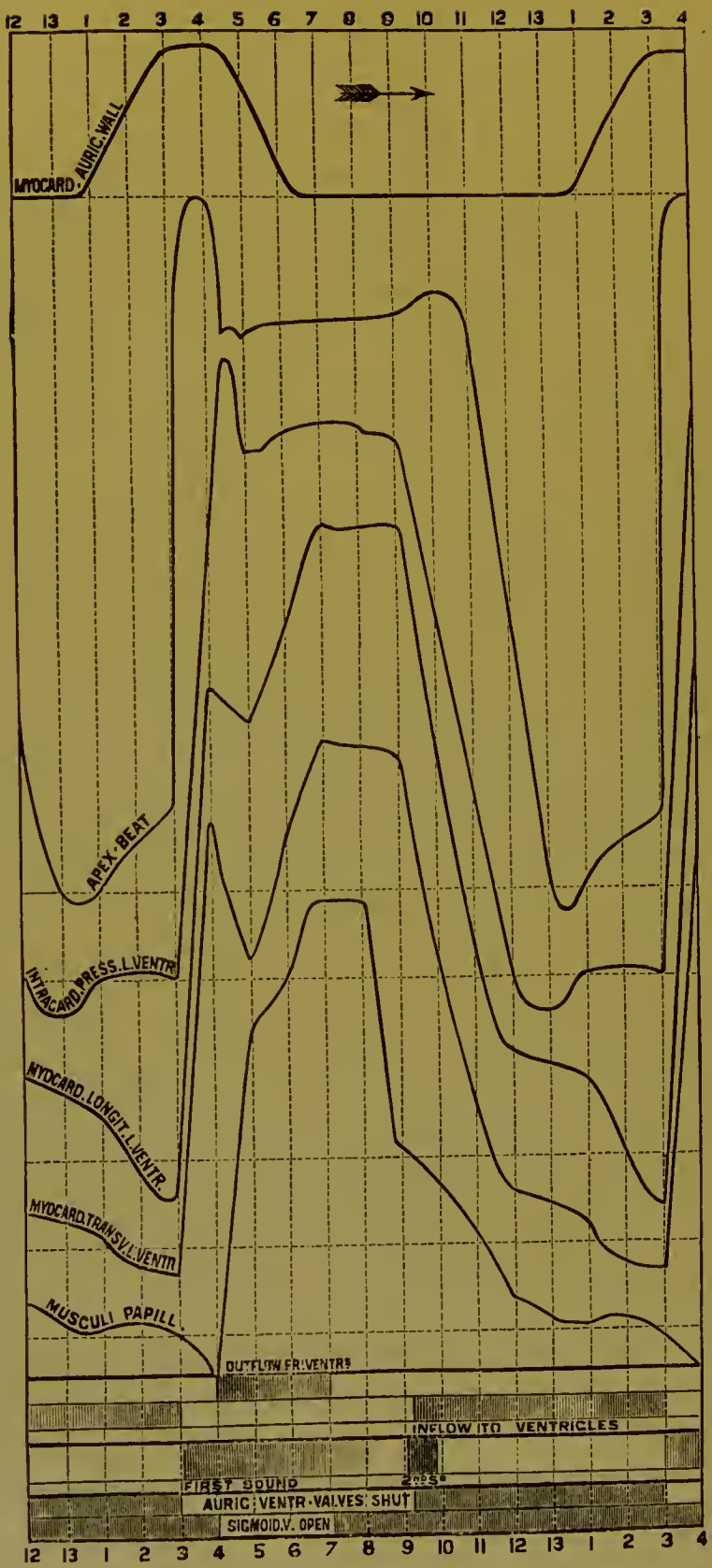
12 13 1 2 3 4 5 6 7 8 9 10 11 12 13 1 2 3 4
MYOCARD. AURIC. WALL
APEX. BEAT
INTRACARD. PRESS. L. VENTR.
MYOCARD. LONGIT. L. VENTR.
MYOCARD. TRANSV. L. VENTR.
MUSCULI PAPILL.
OUTFLOW FR. VENTRS.
INFLOW TO VENTRICLES
FIRST SOUND
2NDS.
AURIC. VENTR. VALVES SHUT
SIGMOID. V. OPEN
12 13 1 2 3 4 5 6 7 8 9 10 11 12 13 1 2 3 4

Fig. 17.

of the apex-beat or of the intraventricular pressure-curve to which we have referred on p. 169. The time of the second sound of the heart can be learned exactly from the position of certain small vibrations on the pulse-curve at the root of the aorta, while the first sound, it need hardly be said, has been shown by others to correspond to the ventricular contraction.

The position of the auricular contraction and expansion we obtained from simultaneous myographic tracings of the auricular and ventricular walls. The other curves on the diagram are those which have been already described.

After spending much time in constructing this diagram and measuring out many tracings, in the fear that preconceived theories as to how the various events of the heart-beat *must* hang together might lead us to disregard facts, we are still by no means satisfied that it is not as likely to mislead as to instruct the reader. If the impression be left in his mind that the heart-beat *always* takes place after the manner indicated in the diagram, so long as the heart and circulation are normal, we must clearly say that such an impression will be erroneous.

The heart is not a machine like a steam-pump, the movements of every part of which must conform with those of the other parts in rapidity and duration. If the oscillations of the piston of such a pump become quicker or slower, the quantity of water pumped out rises or falls in the same proportion. In the case of the heart, however, the amount of work done by the organ, in the way of pumping out blood, bears no constant relation to the rate of the heart-beat. That the characters of the heart-beat are capable of being varied within physiological limits, by a number of influences, will perhaps best be understood by analysing the most important of them one by one. This, moreover, is a subject of great importance to the pathologist, seeing that it is only by a clear comprehension of the variations which the heart-beat is capable of undergoing within normal limits that its mechanism in disease can be understood. It is necessary, in seeking to understand the heart-beat in diseased conditions, to keep in mind that no sharp line of demarcation can be drawn between the action of the healthy, and that of the diseased organ. Any abnormalities of the function of the heart, and indeed of any organ of the body, can only be due to

increase or diminution of some one or more of the factors by which the action of the organ is normally carried on. The variations in the action of the heart, by influences which do not exceed those which occur in health, are those which are of special interest to pathologists, seeing that they verge insensibly into the extremes which are met with in diseases of the organ. Moreover, the continued action of physiological influences, if they be extreme, may lead to conditions which cannot be distinguished except by their etiology from pathological conditions. As an example of this we may refer to the increased size of the soldier's heart after a severe campaign, or to the blacksmith's heart. These enlargements differ in no respect from the hypertrophy of the heart, in, for example, Bright's disease or aortic stenosis, except that they are the direct result of extreme physiological conditions which have been in existence for an unusual time.

Many other examples might be given of such overlapping of healthy and diseased conditions. Take for instance the effect on the heart-beat of changes in the pressure against which it has to empty its contents, in other words the hydrostatic pressure within the aorta and pulmonary artery. It need hardly be said that the blood-pressure in the systemic arteries is capable of varying within fairly wide limits. At each respiration there is a rise and fall, while with muscular exertion, and a variety of other causes which need not here be detailed, still more considerable physiological variations may take place. Still greater variations of the blood-pressure are of course met with when the conditions deviate from the typically healthy condition; speaking roughly it may be said, that the pressure in the systemic arteries may rise as high as 250 mm. of mercury, or more, and may fall as low as 50 mm. without death or syncope from cerebral anæmia taking place.

HEART-BEAT AND PULSE-WAVE.

BY C. S, ROY, M.D., F.R.S.,

Professor of Pathology,

AND J. G. ADAMI, M.A., M.B.,

Demonstrator of Pathology, in the University of Cambridge.

[From the *Cambridge Pathological Laboratory.*]

SECTION V.—Continued.

We have said that the action of the heart is profoundly influenced by changes in the pressure within the arteries. Let us see what is the nature of the modifications so produced, and consider, first of all, how the heart-beat is affected when the arterial pressure rises. We must mention that we do not refer to changes in the rate of the heart-beat which are produced by rise or fall of the aortic pressure (through the medium of the vagus nerves), but to those changes in the character of the individual beat which are independent of the rapidity of rhythm.

One of the effects of rise of pressure in the systemic arteries is to diminish the extent to which the fibres of the heart-wall are shortened during systole; this diminished shortening being identical in nature with the diminished degree of shortening of a voluntary muscle when in an experiment the weight which it has to raise is increased. The effect of this diminished shortening is, as we have shown,[1] to increase the quantity of residual blood which is left in the ventricle at the end of systole. This increase in the residual blood does not, however, under normal

[1] Roy and Adami, *British Medical Journal*, December 15, 1888.

conditions, lead to a diminution of the amount of blood expelled by the heart in a given time, seeing that it is compensated for by an increased expansion during diastole: in other words, *rise of the arterial pressure leads to physiological dilatation of the ventricles*, which may, as we have shown elsewhere, in extreme cases give rise to functional incompetence of the auriculo-ventricular valves. We may mention that functional incompetence of the same kind may result not from an increase in the work which the organ has to do, but from diminished power of contraction, the result of impaired nutrition or disease of its walls. We repeat, however, that in strictly healthy conditions any rise of the blood-pressure produces a corresponding physiological dilatation of the heart. Pathological dilatation, resulting in functional incompetence, differs in no respect save in degree from physiological dilatation.

Another important effect of continued high arterial pressure, where the conditions are favourable, is hypertrophy of the ventricular walls; but as we think it better to confine our remarks in the meantime to the beat of the unhypertrophied heart, we shall say nothing here upon this subject.

Of more direct interest in the present connexion is the effect of rise or fall of arterial pressure upon the parts played by the contraction of the ventricular wall and the *musculi papillares* respectively, in their common function of expelling blood from the ventricle. As we have shown in Section III., the superposed wave between 4 and 5 of the intracardiac curve in Fig. 17, which ordinarily constitutes the highest pressure produced in the heart during systole, is due to the contraction of the papillary muscles. When, however, the arterial pressure is raised, the height of this wave above the rest of the curve is lessened, and the effect is that with a certain arterial pressure this wave does not rise so high as the rounded prolonged shoulder which follows it; in other words, the intracardiac pressure curve has a notch on the ascending line, that is to say, is anacrotic. (This term is usually given to curves with a notch on the ascending line; dicrotic being a term used to describe a curve with a notch on the descending line.) It must be understood that this change in the character of the intracardiac pressure curve is produced by alterations of the mean pressure that are within physiological

limits; in other words, the anacrotic curve is a normal modification of the usual form as described in Section III. The explanation of this change in the form of the intraventricular pressure curve as a result of a rise of arterial pressure is important, seeing that upon it also rests the explanation of the anacrotic *pulse* wave. There are certainly two factors, and probably a third, which take part in this change in the intracardiac pressure-tracing, where the arterial pressure is raised. Let us consider these *seriatim*.

In the first place, the heightened pressure upon the arterial side of the sigmoid valves delays the opening of these, which can of course only take place when the pressure in the ventricle is raised so as to be higher than that in the aorta (or pulmonary artery in the case of the right ventricle). As, however, there is no corresponding delay in the contraction of the *musculi papillares*, a smaller portion of the papillary muscle contraction is utilised for the purpose of expelling the blood. Seeing that there is no diminution in the total volume thrown out at each systole, this leaves a larger portion of blood to be expelled by the continued contraction of the heart-wall after the auriculo-ventricular valves have been pulled down by the first rapid and effective part of the contraction of the papillary muscles. In other words, a rise of pressure in the arteries diminishes the amount of blood thrown out by the contraction of the papillary muscles, and increases that thrown out by the heart-wall.

This change in the amount of blood expelled, as the result of the contractions of the papillary muscles and heart-wall respectively, is perhaps exceeded in importance by the second factor, which tends when there is high arterial pressure to render the intracardiac pressure curve anacrotic.

This second factor arises from the characteristics of the arterial wall as regards elasticity. As has been shown by one of us elsewhere,[1] the arterial walls become more rigid as the pressure is raised above the general mean pressure of the animal from which the arteries are taken. This greater rigidity tends to raise the pressure towards the end of systole, the curve in other words tending to become, or becoming, anacrotic.

As it is desirable that this point should be clearly understood,

[1] Roy, *Journal of Physiology*, vol. iii.

we will describe an experiment by which the relation which normally exists between the cubic capacity of a piece of artery and the intra-arterial pressure may be easily demonstrated.

From a freshly-killed animal, say a rabbit, a short length of any artery is taken, and one end being ligatured the other end is tied on a cannula of appropriate size. The cannula is connected by means of a T tube, on the one hand with a mercurial manometer, and on the other hand with a syringe by which air may be injected into the bit of artery. The latter is placed in a rigid-walled box of any convenient shape containing olive oil, and is connected with an arrangement for recording the amount of oil which is forced out of the box by any increase in its cubic dimensions.

In Fig. 18 is reproduced a graphic record from an experiment with the above-described apparatus. It was obtained from the carotid of a rabbit.

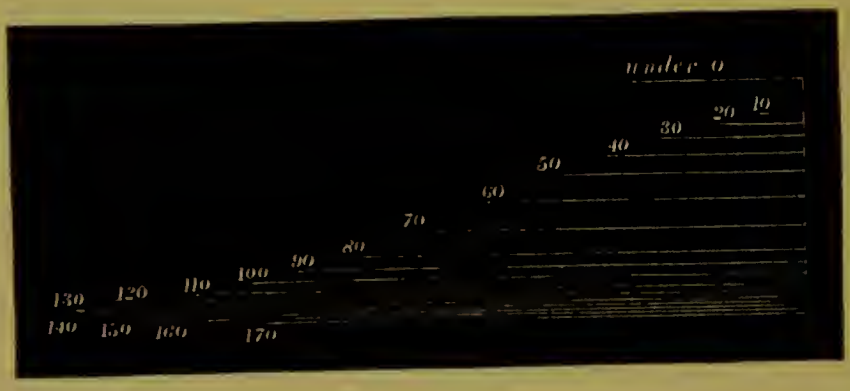

FIG. 18.

Tracing showing relation between intra-arterial pressure and cubical contents of normal artery. The figures give pressures in millimetres of mercury, while the distance of the horizontal lines from the uppermost corresponds to the cubic capacity of the vessel. *N.B.—The length of the horizontal lines varies simply in order that the pressure with which they correspond may be clearly indicated.*

The upper horizontal line (drawn, like the others by moving the recording cylinder backwards and forwards with the hand) represents the cubic dimensions of the piece of arterial tube when collapsed, owing to the pressure outside its wall being higher than that within. On now raising the intra-arterial pressure until the mercury stands at 10 mm., the vessel opens out and expands slightly, the increase in

its contents being represented by the distance between the upper and next highest horizontal lines. On increasing the pressure to 20 mm., the cubic capacity of the vessel is increased to an extent corresponding to the distance between the lines marked 10 and 20 respectively. The other horizontal lines represent the cubic capacity of the artery at the pressures marked at the end of each line.

The tracing shows—that the increase in volume with each successive increment of pressure becomes greater till about 60 mm. of mercury is reached ; that the maximum increase is between 60 and 80 mm., and that with higher pressures the increments in cubic capacity become less and less, so much so, that raising the intra-vascular pressure from 110 to 170 mm. produces only about an equal increase in contents to that produced by raising the pressure from 60 to 70 mm. Now 70 or 80 mm. of mercury is about the normal mean blood-pressure of the rabbit, and this experiment shows that above this the arteries become more and more rigid-walled.

In considering the question of the mode of production of the anacrotic pulse, it must be remembered that a rise of blood-pressure within normal limits does not diminish the amount of blood expelled at each contraction—that, on the contrary, the slowing of the heart which usually accompanies rise of blood-pressure results in a corresponding increase in the volume thrown out at each systole. From the tracing (Fig. 18), it can be seen at a glance what would be the increments of pressure within the artery if a given volume of blood were forced into it at 60 mm. and 110 mm. respectively—it can be seen that they would be in the proportion of about 1 to 6. That this great rise with high pressures must tend to make the pulse and intra-ventricular pressure curves anacrotic, *i.e.*, rising towards the end of the period of outflow from the heart, is evident enough.

There is however a *third* influence which in all probability acts in the same way as the two factors which we have just referred to : we refer to the diminished bulging upwards of the auriculo-ventricular valves which must take place when the heart becomes dilated, owing to the increased distance of the point of origin of the papillary muscles from the base of the ventricles. Unless the papillary muscles became stretched to a degree

sufficient to allow the auriculo-ventricular valves being bulged upwards to the same extent as before the physiological dilatation of the heart (which is improbable, seeing that the causes which produce the dilatation of the ventricular walls do not affect the papillary muscles to the same degree), the diminished bulging upwards of the auriculo-ventricular flaps will necessarily lead to the contraction of the papillary muscles being less effective. The effect which their contraction produces upon the pressure curve will therefore be relatively less. In addition to this it must be kept in mind that any distension of the heart-wall will of itself render the papillary muscle contraction less effective, seeing that the part from which the papillary muscle arises will, *cæteris paribus*, be more readily pulled in, so that the contraction force of the papillary muscle will be, in part, expended in distorting the ventricle.

The above considerations explain why it is that the intraventricular pressure curve—and at the same time, the pulse-wave—undergo the characteristic change from the normal to the anacrotic form. We shall have more to say upon this subject when we come to speak of the pulse-wave.

Lowering the arterial pressure below its usual height produces also important changes in the various elements which compose the heart-beat, and which will have to be considered in detail when we come to discuss the pulse of low pressure. It will be as well for us, however, to say something here regarding some of the effects of diminished pressure upon the mechanism of the heart-beat. In the first place, with low arterial pressure the ventricles are more completely emptied at the end of systole than is otherwise the case. Often, moreover, though not always, the low pressure is due to the small amount of blood expelled by the heart at each contraction. Both of these factors tend to make the papillary muscle contraction play a more prominent part in raising the intra-ventricular pressure and expelling the blood than it would otherwise do. The low pressure in the arteries permits the opening of the sigmoid valves at a comparatively early period of systole, while the small volume of blood to be expelled, and in any case the small resistance offered by the arteries to the inflow of blood into them, cause the outflow from the ventricles to be concluded at a com-

paratively early period of systole. In illustration of this we give Fig. 19, which shows the curve of contraction of the ventricle-wall and of the apex-beat with low arterial pressure. It can be seen that the actual shortening of the fibre of the heart-wall occupies a smaller proportion of the whole systolic

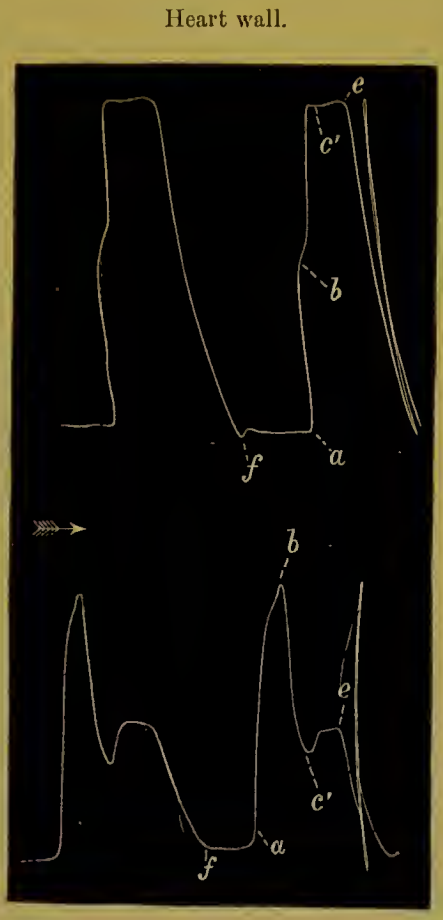

FIG 19.

Apex-beat. Myocardiographic and apex-beat curves taken with sub-normal arterial pressure.

period than is the case with higher arterial pressure, as for example, in Figs. 2 and 5 on pp. 85 and 90 respectively. The apex-beat curve shows the large diminution of diameter that takes place during the time (*b* to *c'*) of the contraction of the papillary muscles. The heart-wall curve shows that the period

of active shortening is diminished, while the succeeding period of persistent contraction is increased in duration.

The effect of this shortening of the period of outflow, and earlier commencement and ending of it, is necessarily that the pulse-wave is made up mainly by the superposed wave of the intracardiac pressure-curve, which is due to the contraction of the *musculi papillares*. This again is a matter which can be better discussed under the heading of the form of pulse-wave.

Increased quantity of blood passing through the heart is also a matter which can best be discussed in connexion with the pulse.

The heart-beat is modified in certain important particulars by the nerves supplying it. As, however, these nerves affect the ventricles less than the auricles, and as to describe the effect of these nerves upon the heart would lead us too far, we prefer to say nothing here of their action on the heart.

Changes in the rate of beat also produce modifications, the most important of which is that, *cæteris paribus*, the faster the rhythm the smaller the amount of blood thrown out by the ventricles at each contraction. We cannot, however, go into this subject here.

Of greater importance in the present connexion as regards the mechanism of the heart is the relationship in force and in rhythm of the heart-wall and papillary muscles respectively. It would naturally be assumed *a priori* that, these two parts of the heart being so closely allied one with another, the force of contraction of the one is incapable of being varied independently of that of the other. Still less would it be expected that the rhythm of the two might be to a certain extent independent. Such, however, is the case, as can be shown without difficulty by the graphic method employed by us and described in our first two Sections.

In the first place, the force of the contraction of the *musculi papillares* may vary independently of that of the heart-wall. A very good example of this is seen in the influence upon the heart of the tincture obtained from the seeds of *Strophanthus hispidus*.

If, having arranged the myocardiograph so as to obtain simultaneous tracings of the contractions of the heart-wall and

of the papillary muscles, and having reduced the rapidity of revolution of the drum upon which the tracings are being taken, so as to be able to follow the changes in height of these two tracings for as long a time as is desired, we then inject into a vein a maximum medicinal dose of the tincture, the effect of the drug shows itself very rapidly. The first change noticed is great increase in the force of the contractions of the *musculi papillares*, without any or with only very slight increase in the force of the contractions of the ventricular wall. This change is the cause of the increased efficiency of the papillary muscles in raising the height of the superposed wave of the intra-cardiac pressure-curve. This can be seen in curve B, Fig. 7, p. 163. We must not be understood to imply that this is the only effect upon the heart of medicinal doses of strophanthus; it is, however, apparently a very important, if not the most important, action of the drug, and serves to explain why it is that in certain forms of heart-disease strophanthus is so valuable a remedy, whilst in others it produces little if any benefit. Knowing this action of strophanthus in increasing the force of the papillary muscles we would not expect it to be of much use in cases of mitral stenosis, especially if, as is often the case, there be marked rigidity of the mitral flaps and atrophy of the musculi papillares. On the other hand, in cases of functional incompetence of the mitral or tricuspid from whatever cause, we would expect that advantage, and in some cases very great gain, would result from the employment of this remedy. This we believe corresponds fairly well with clinical experience, although of course in cases where the papillary muscles have undergone extensive fatty degeneration, or sclerosis, or other form of regressive metamorphosis, it is not to be expected that so much benefit can result from the action of the remedy in question as in cases where the muscles have undergone no important anatomical change.

If in our experiment a second dose of strophanthus be injected shortly after the one previously mentioned, the toxic effects of the drug show themselves. One of the most striking of these is the weakening of the contraction of the papillary muscles, which rapidly replaces the increased force that was so marked a result of the former dose. There is also a slight weakening of the heart-wall contractions, but this is very much

less marked than the corresponding effect upon the papillary muscles. One effect of this weakening is to lower the height of the superposed wave of the intra-cardiac pressure curve, as is well seen in curve D, Fig. 7, p. 163. *This weakening may go on to complete arrest of the contractions of the papillary muscles*, the ventricular walls continuing to contract. Before ceasing, however, it is by no means rare to find want of co-ordination between the papillary muscle contractions and those of the heart-wall. Some of the latter are not accompanied by contractions of the papillary muscles, while others are: on the other hand the papillary muscle may show two contractions for one of the ventricle wall. It need hardly be said that these facts render it probable enough that in certain diseased conditions of the heart a similar want of co-ordination may present itself.

Besides strophanthus, other causes, we find, may produce want of co-ordination between these two elements of the ventricular contraction. For example, in some cases, temporary arrest of the artificial respiration causes complete, or nearly complete, arrest of the papillary contraction. We have reason to believe that many other causes besides those just mentioned affect these two elements unequally.

We are tempted to conclude this Section by stating the results of our observations upon the various forms of irregularity of the heart, but the subject is too large, and its discussion would lead us more deeply into the physiology of the heart than is here desirable.

It is perhaps as well for us to state that we do not for a moment consider that we have given a complete account in this Section of the mechanism of the heart-beat; our object having been merely to refer to those matters which are of more immediate importance in connexion with the pulse.

SECTION VI.

THE NORMAL PULSE-WAVE.

When Marey's sphygmograph was first introduced it was not unnaturally anticipated that it would be found of great practical value. This anticipation has not, it must be confessed, been

realised; and it may indeed be questioned whether any data of practical value regarding the pulse obtainable by the sphygmograph cannot be arrived at just as satisfactorily and very much more easily by the educated touch. It is of course capable of giving a permanent record of certain characters of the pulse-wave; but we think it open to doubt whether it has yielded anything more than this to the vast majority of those who have employed it. Nevertheless it would appear that in the hands of some few physicians who have attained great skill in the application of the instrument, it does give information which is of practical value and which cannot otherwise be obtained. The curves published by Mahomed, Lorain, Riegel, and one or two others are evidence of this. On the other hand, there have been physicians of unquestioned ability who have devoted much time and care to the employment of the sphygmograph at the bedside, and who have not found that much, if indeed anything, is to be gained by its use. Dr. Broadbent, for example, in his valuable monograph upon *The Pulse*, recently published, has but little to say regarding the advantages of using the sphygmograph rather than the educated touch.

The reason of this comparative failure of the sphygmograph for clinical observation is not, we think, very far to seek; it consists, we believe, in the fact that the instrument itself is faulty; we cannot for example be sure of getting the same form of pulse-wave on two successive applications, although there is no reason to suppose that in the interval the form of the pulse has undergone any change. We have no adequate means of controlling the pressure of the button on the artery, nor can we always be certain that the button occupies the same position over the vessel. We might give other objections to the instrument, did we think that its faults were insufficiently recognised. Nor do we consider that the modifications which the instrument has undergone at the hands of various observers have removed the various objections to it; while one of these modified sphygmographs is, we think, very much less satisfactory than Marey's original instrument. We refer to Dudgeon's sphygmograph, tracings from which are so frequently disfigured by inertia-vibrations as to render the curves obtained from it more or less

worthless. The mechanical construction of this instrument is such as to render great inertia-vibrations unavoidable.

It must not be understood from the above that we think it impossible to obtain sphygmographic tracings which will give information of value unattainable by the trained touch of the physician, All that we wish to say is that the instruments at present in use are extremely unsatisfactory.

To obtain satisfactory graphic records of the pulse an instrument is required which will give with certainty similar results when applied at different times, when the pulse has not undergone alterations in the intervals; which, secondly, can be applied without difficulty; thirdly, the pressure upon the artery must be capable of being regulated with exactitude; fourthly, it must give tracings sufficiently large to enable us to distinguish minute variations in the characters of the pulse-wave; fifthly, the curves obtained must be free from inertia-vibrations; sixthly, it must be capable of measuring the blood-pressure within the artery; and lastly, it must be such that it can be conveniently employed at the bedside.

We have been employing an instrument which, in our opinion, fulfils these requirements. The principle of its construction is to cover over and enclose a portion of the radial artery in an air-tight rigid-walled box, the radius forming one side. The box is filled with water, the pressure of which can be varied at will, and which conveys to a piston the expansion and contraction of the artery at each pulse-wave, the movements of the piston being communicated to a recording lever.[1] In Fig. 20 are given examples of pulse-tracings obtained by this instrument from one of our own wrists, the pressure upon the artery being 30, 45, and 70 mm. of mercury respectively. In Fig. 21 we give a pulse-curve obtained from the unopened femoral artery of the dog by a method similar in principle to that just referred to. Fig. 22 shows a tracing from the root of the ascending aorta of a dog, the thorax having been opened. This was obtained by a light recording lever connected with a button which was pressed

[1] Although this instrument is at present in a condition which allows of its being used at the bedside, it is thought preferable to wait before giving a detailed description of it uutil by further experiments its most convenient form has been decided. Such a description will be given before the conclusion of these articles. —C. S. R.

upon the vessel by a spring of india-rubber thread, the part of the aorta from which this was taken being supported below by a flat

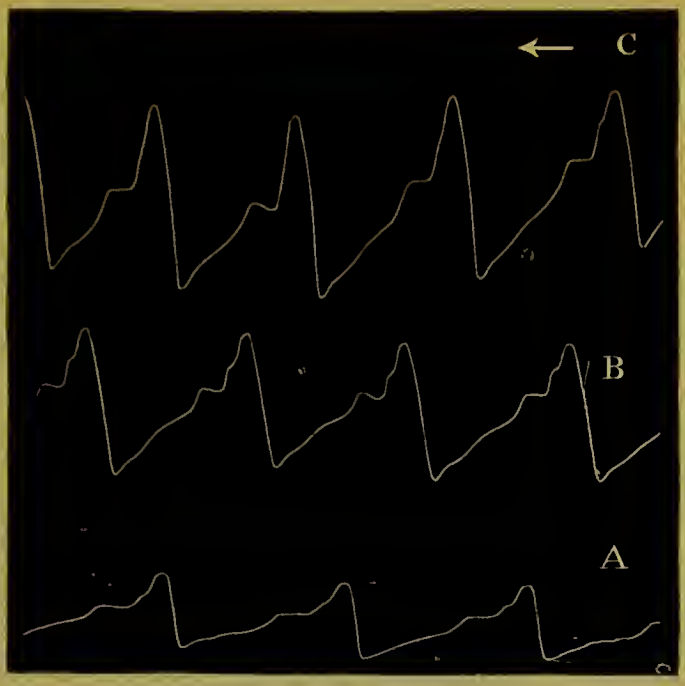

FIG. 20.

Normal pulse-tracings from human radial of healthy male adult.
A, with extravascular pressure of 30 mm. of mercury.
B, ,, ,, ,, 45 ,, ,,
C, ,, ,, ,, 70 ,, ,,

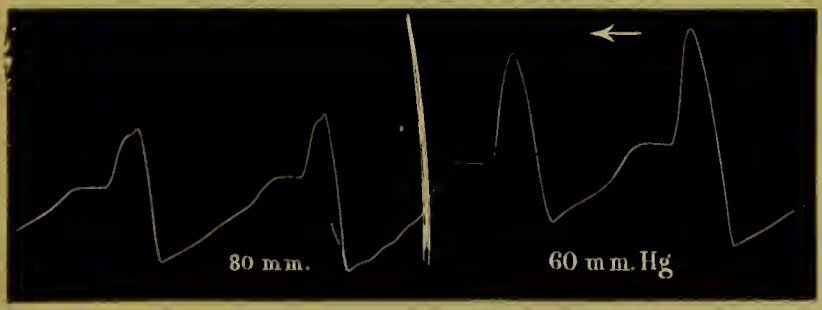

FIG. 21.

Pulse-tracing from femoral of dog, obtained by a method similar to that employed in Fig. 20.

metal hook. In Fig. 23 are tracings taken from the carotid of a rabbit by a method described by one of us,[1] which consists in

[1] Roy, *Journal of Physiology*, vol. ii. p. 66.

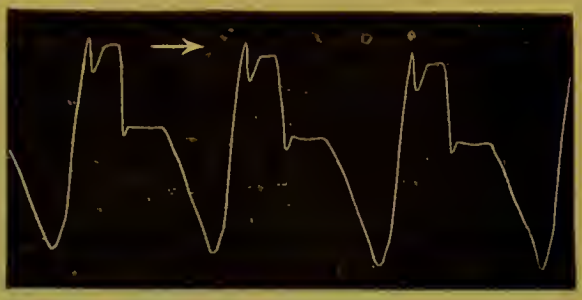

FIG. 22.

Pulse-tracing from the root of the ascending aorta of a dog, obtained by a method similar to that of the ordinary sphygmograph.

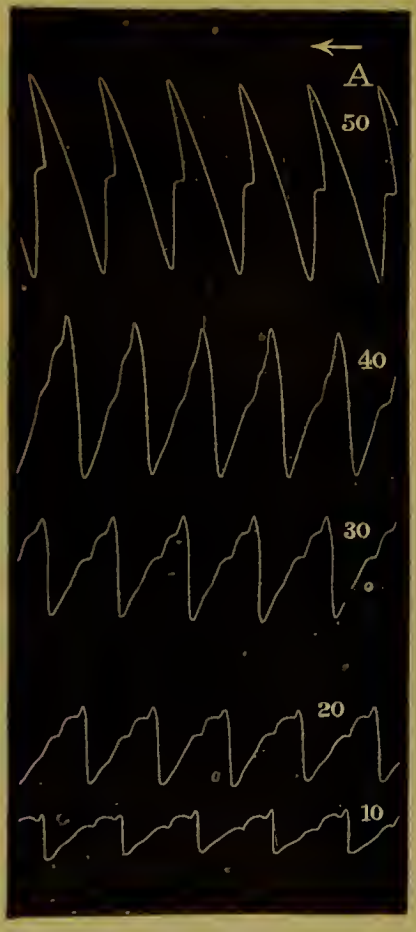

FIG. 23.

Pulse curves from carotid of rabbit with extra-arterial pressures of 10, 20, 30, 40, and 50 mm. of mercury.

enclosing a piece of the unopened artery in a rigid-walled box, the expansion and contraction of the artery at each pulse-wave being recorded by a piston-lever arrangement. The pressure upon the artery could be varied as desired, and the figure shows tracings obtained with extra-arterial pressures of 10, 20, 30, 40, and 50 mm. of mercury.

These different pulse-tracings from man, the dog, and the rabbit obtained by various ways resemble one another in their chief characteristics. They all show an uninterrupted and more or less rapid upstroke to the culminating point of the curve, which forms the apex of the first secondary wave of the pulse; between this and the next small wave there is a notch, generally known as the predicrotic notch, while after the second secondary wave comes the deeper dicrotic notch. This is followed by a more or less prominent short wave, between which and the lowest point of the curve there is a long flattened wave. These then, we may take for granted, are normal characteristics of the pulse-wave, seeing that they appear in sphygmographic tracings taken from different arteries of different animals and by different instruments.

HEART-BEAT AND PULSE-WAVE.

BY C. S. ROY, M.D. F.R.S.,

Professor of Pathology,

AND J. G. ADAMI, M.A., MB.,

Demonstrator of Pathology, in the University of Cambridge.

[From the *Cambridge Pathological Laboratory.*]

SECTION VI.—Continued.

THE NORMAL PULSE-WAVE.

We have now to consider what is the meaning of each of the chief features of the normal pulse-curve. With regard, first of all, to the up-stroke: it is of course due to the correspondingly rapid rise of pressure in the artery, which commences at the root of the aorta at the moment of opening of the aortic valve. This is opened when the pressure within the ventricle exceeds that within the aorta. If the mean pressure in the aorta be high, the valve opens at a later period of the cardiac systole than would otherwise be the case, and *vice versâ;* on the other hand, the quantity of blood in the heart at the end of diastole, and the force of the ventricular contraction, influence the rapidity with which the intraventricular pressure is raised during the earlier part of systole, and in this way also may cause a change in the time-relation between the commencement of the ventricular contraction and the opening of the valve.

It must be kept in mind then that at the root of the aorta the beginning of the pulse-wave does not bear a constant relation in time to the beginning of the ventricular systole, a fact which is of considerable importance in influencing the form of the pulse-wave.

With regard to the height of the up-stroke, this depends, *cæteris paribus*, on the extent of the rise of pressure which is produced in the arteries by the inflow of blood from the ventricle. The extent of this rise is influenced by a variety of causes, namely (1) by the volume of blood expelled by the ventricle, (2) by the freedom of outflow of the blood into the capillaries, and (3) by the degree of rigidity of the arterial walls, which, as we have shown, varies with different arterial pressures. Besides these, the height of the pulse-wave as shown in the sphygmographic tracing is very greatly influenced by the pressure of the recording instrument upon the vessel, as can be seen upon reference to Figs. 20 and 23 (pp. 359 and 360). This is a matter which presently we shall have to discuss in some detail. We may therefore pass it by in the meantime.

The rapidity of the up-stroke—that is to say, the time which is occupied by the rise of pressure (and which can be ascertained by measuring the distance of the top of the curve from a line drawn by the recording lever through the point of commencement of the up-stroke when the clockwork is at rest)—varies also with the rapidity of outflow from the heart. But into this question we cannot enter until we have considered the meaning of the superposed waves, and especially of the first of these.

It is of special importance to know what point upon the sphygmographic curve corresponds to the cessation of outflow from the ventricle. This it is perfectly easy to determine by taking simultaneous tracings of the pulse-beat at the root of the aorta, and myographic curves of the ventricular wall, the latter showing the moment when the shortening of the fibres of the myocardium is completed. This point, it need hardly be said, must be that of the cessation of outflow. Fig. 24 gives two curves obtained in this way from the heart of a dog, which have been carefully measured out so as to obtain their exact relations in time. The figures on the two tracings mark points which are synchronous. It can be seen that the rise of pressure in the aorta begins after the heart-wall has performed a certain part of its shortening, and that the point 4, which is placed on the ventricle-wall curve at the moment of cessation of *shortening* (though not of *contraction*), is represented upon the

pulse-curve by the commencement of the rapid descent which precedes the dicrotic notch. It is only however in cases where the cessation of the outflow is fairly abrupt that this point is recognisable upon the pulse-curve. Where the outflow ceases gradually, as indeed is ordinarily the case, there is no point upon the pulse-tracing which can be recognised as synchronous with the arrest of the outflow. In any case this must precede the dicrotic notch.

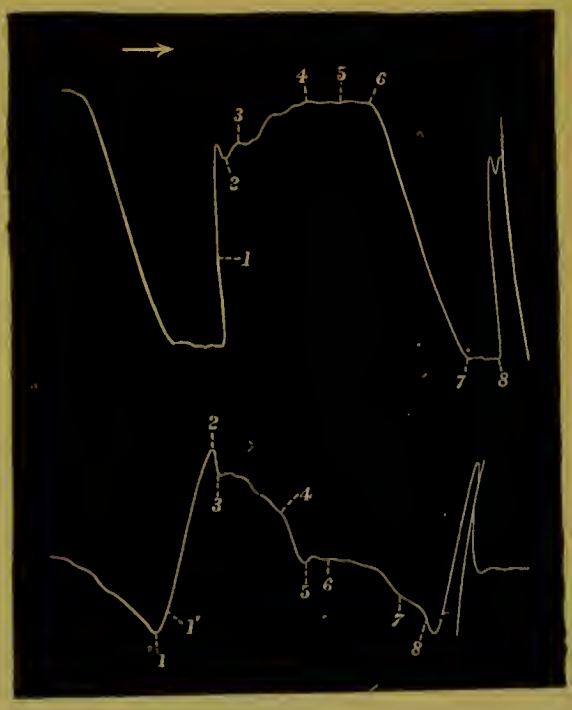

FIG. 24.

Myocardiographic tracing (upper curve) from left ventricle (longitudinal) of dog, taken simultaneously with pulse-tracing (lower curve) from root of aorta. The numbers show synchronous points.

Between the commencement of the up-stroke and the dicrotic notch there are two waves, sometimes called the *percussion* and the *tidal* wave respectively. What is the cause of these? Their nature has long been a matter of discussion, and various investigators have sought to find their meaning by experiments with more or less ingenious artificial *schemata*, consisting of some form of pump and a system of elastic tubes. We must admit that we have little confidence in the results of such imperfect imitations of Nature, and do not think it profitable for us to criticise in

detail either the methods used or the results obtained in such experiments.

As we have pointed out, there is on the intraventricular pressure-curve, and during that part of it which corresponds to the time when the aortic valve is open, a superposed wave

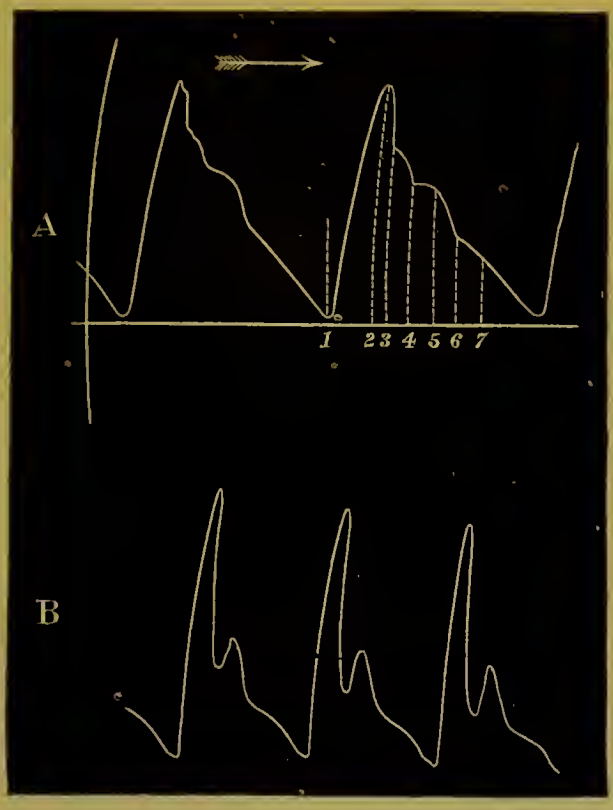

FIG. 25.

A, normal pulse from radial of healthy adult male, obtained by *sphygmometer*. Extra-arterial pressure of 70 mm. of mercury. 1 to 2, up-stroke ; 2, apex of papillary wave ; 3, notch between papillary and outflow-remainder waves (predicrotic notch) ; 3 to 4, outflow-remainder wave ; 4, dicrotic notch ; 4 to 6, post-dicrotic wave ; 6, post-dicrotic notch.

[*Note.*—The small notches on the first and second of the superposed waves of the first of these pulse-curves have crept in by mistake in the engraving, and are not present in the original curve.]

B, from same artery with same extra-arterial pressure, taken during acute catarrh of upper respiratory passages.

due to the sudden pulling down of the auriculo-ventricular valves by the papillary muscles, during the first rapid part of their contraction. It is easy to prove that this superposed

wave, of the intraventricular pressure-curve, corresponds exactly in time with the first superposed wave of the pulse-curve—the one which usually forms the culminating part of the tracing.

In Fig. 24 it can be seen that the arrest or slowing of the shortening of the ventricular muscle, which results from the rise of pressure due to the contractions of the papillary muscles, is synchronous with this first superposed wave of the pulse-tracing. In other words, *it is due to the contraction of the papillary muscles.* We must therefore reject the name *percussion wave* which has been given to it, and will henceforth refer to it as the first superposed wave of the pulse-curve, or, for shortness' sake, as the *papillary wave.* The next of the secondary waves of the pulse, that, namely, between the papillary wave and the dicrotic notch, corresponds with the rounded shoulder of the intracardiac pressure-curve (*e*, Fig. 7, A and B, p. 163). In other words, the pulse-curve, during that part of it which corresponds in time with the outflow from the ventricle, agrees in form with the intraventricular pressure-curve. As we have shown, the rounded shoulder is due to the continued contraction of the heart-wall and papillary muscles after the flaps have been pulled down. This second wave, which is usually known as the tidal or predicrotic, may therefore be termed the *outflow-remainder* wave. It corresponds in time with our third phase of ventricular systole (p. 93).

After this wave comes the dicrotic notch. The usual explanation of this negative wave is that it is due to the inertia of the blood in the aorta and larger arteries, which has gained a certain velocity during the period of outflow from the ventricle, and which upon the blood ceasing to leave the ventricle necessarily causes a negative wave, commencing at the root of the aorta, and propagated in the same way as the positive wave. This explanation is, so far as we know, the correct one. As might be expected, we find that any cause which increases the abruptness of the cessation of the outflow, or increases the velocity with which the blood is expelled from the heart, tends to make this notch more pronounced; on the other hand, the pressure in the arteries is also of influence upon the depth of this negative wave, for the simple reason that the more rigid the elastic walls of the arteries the more readily will such a (negative) inertia

wave result at the termination of the inflow through the aortic valves.

As we have pointed out in our last Section, the arterial walls are very much more rigid with high pressures than with pressures at or below the normal; in other words, the conditions, in so far as the elasticity of the arterial walls comes into play, are more favourable for the production of a dicrotic or inertia notch, than when pressures are at or below the normal. This is opposed to the ordinary teaching upon this subject, it being stated that the increased dicrotism which results from loss of blood or in certain stages of fever, &c., &c., is due to the low pressure, which renders the arterial wall more flaccid.

It seems to us curious that such a theory should have been advanced, and still more that it should have been generally accepted, seeing that the one characteristic of the elasticity of the arterial wall which is more fully recognised than any other is, that this elasticity permits of an intermittent current being transformed into a more or less continuous current, and that the more elastic the arterial wall the more perfectly will it carry out this function. This elasticity will at the same time reduce such secondary inertia-waves as that which causes the dicrotic notch. From our present standpoint it is to us surprising that such an evident contradiction should have gained general acceptance, unless indeed it has been due to a want of general recognition of the fact that the arterial wall becomes more rigid with higher pressures.

As we shall have presently to enter into the conditions which lead to the accentuation or the reverse of the dicrotic notch, we will say no more here upon the subject, except that its depth is necessarily influenced by the height of the post-dicrotic wave, and that the height of this does not always depend upon inertia alone. We find, namely, that this positive post-dicrotic wave, when a tracing has been taken at the root of the aorta, not infrequently corresponds exactly with the sudden commencement of the ventricular diastole; and it is very probable that this sudden change in the rigidity of the heart will affect the aorta and its contents through the connexion between the aortic and ventricular walls. In some cases, where the commencement of ventricular diastole does not exactly

correspond with the post-dicrotic or positive inertia-wave, but soccurs a little later, we find that in place of one there are two mall waves following the dicrotic notch; and one of these corresponds in time with the commencement of the diastole.

Following the post-dicrotic (positive) wave when the heart rhythm is not too rapid, there is a low rounded prominence on the line of descent, which, so far as we know, is of the nature of a second positive inertia-wave. To be more exact, we know of no other means of explaining it.

Finally, immediately before the up-stroke, there is often enough, in tracings taken from the ascending or descending thoracic aorta, a small notch followed by a short low positive wave. This does not usually appear upon the pulse at some distance from the heart, and appears to be due to the pull upon the aorta which results from the first part of the ventricular systole, before the aortic valves are opened.

To make the above description of the normal pulse-tracing clear, we may epitomise what we have written above by tabulating the following as its constituent features:—

(1) The *up-stroke*, which may vary in rapidity of rise and in height (1 to 2, Fig. 25).

(2) The *papillary wave*, or first secondary wave, sometimes called the percussion wave, the height of which, as compared with the succeeding secondary wave, corresponds to the distance between the top of the up-stroke and the pre-dicrotic notch (2 Fig. 25).

(3) The *outflow-remainder wave*, or second secondary wave, usually called the tidal or pre-dicrotic wave (2 to 3). During this wave the outflow from the ventricles terminates, and the cessation may or may not lead to the appearance of a sharp angle before the dicrotic notch, according to the suddenness with which the outflow ceases, &c.

(4) The *dicrotic notch*, the depth of which is measured by the vertical distance between the bottom of the notch and the top of the post-dicrotic wave. This is due to the inertia of the blood.

(5) The *post-dicrotic wave*, due to inertia, the form of which can however also be affected by the sudden relaxation of the ventricular wall at the end of systole.

(6) The *rounded shoulder* which lies between the post-dicrotic wave and the lowest point of the tracing.

(7) Finally, in tracings taken near the heart, a small *notch* and short *positive wave* corresponding in time with the commencement of the ventricular systole.[1]

SECTION VII.

CONDITIONS LEADING TO INCREASED DEPTH OF THE DICROTIC NOTCH.

General Considerations.

It would be very desirable in many respects to have names which describe accurately the various forms of pulse-wave. Such a nomenclature would make it very much easier to write about them. The difficulty however of finding words which will correctly express the manifold characteristics of the complicated curves which are obtainable from the pulse is so great, that we believe it to be practically insuperable. We at all events are not in a position to introduce any satisfactory system of nomenclature for the different forms of pulse-wave, although of course words have been introduced which in some cases, regardless of cacophony, do describe certain features connected with pulse-tracings. Even such general simple words as dicrotic and anacrotic—the one indicating that the pulse has a notch on the down-stroke, the other on the up-stroke—are, in our experience, exceedingly likely to confuse. To some, the application of a name to any given characteristic of the pulse-wave gives to that characteristic a prominence and importance which it by no means necessarily merits, and leaves on the mind

[1] It may be noticed that we have only spoken of two secondary waves before the dicrotic notch. We are aware, however, that in pulse-tracings taken from the dog, with certain pressures of the button on the aorta, three such waves can be distinguished. But in most cases the second and third waves run so much into one another, and are so nearly equal in height that, as can be seen in Fig. 22, they look more like one wave than two. We are inclined to look upon the second of the three, when three are present, as being an inertia-wave following on and due to the rapid fall of pressure between the apex of the pulse-wave and the pre-dicrotic notch. In the human pulse, and in the dog's taken at some distance from the heart, only two waves precede the dicrotic notch. We have therefore not thought it worth while to dwell upon the matter.

on that account an erroneous impression of the characteristic to which it is applied.

We are of opinion that the sphygmographic curve describes its own characters, and the attempt to describe these in words with any brevity, is, we think, manifestly vain.

We are here concerned more especially with the variations in depth of the dicrotic notch, regarding which the inadequacy of our present nomenclature is very apparent. The ordinary healthy pulse has a notch on the down-stroke, and may therefore correctly be termed a dicrotic pulse. Some writers however really mean, by a dicrotic pulse-wave, one in which the notch is deeper than usual; while some confine this name to those cases where the notch can be felt with the fingers; some again give a special meaning to such terms as "fully dicrotic" and hyper-dicrotic; and in short there is a good deal of confusion in the matter. Let us begin by saying, first of all, that the depth of the dicrotic notch varies in different individuals, and in the same individual under different physiological conditions; and when in the following paragraphs we use the term "usual depth" of notch we refer to that found, as far as we know, in all healthy individuals, the tracing being taken while the person is sitting as quietly as possible, or better still, when the breathing is momentarily interrupted, the glottis however being open. In such tracings there is little or in most cases no real rise of the lever point after the dicrotic notch, the level of the highest point of the post-dicrotic wave scarce rising above that of the dicrotic notch. This, so far as dicrotism is concerned, is the typically normal pulse-wave, though it must be clearly understood that extreme degrees of depth of the notch may show themselves temporarily in perfectly healthy individuals. This can be seen in Fig. 26, taken from the radial artery of one of us. During the first part of this curve there was high intra-pulmonary pressure, as is shown by the upper of the two horizontal lines, which gives the level of the mercury in one limb of the manometer connected with the mouth. The actual intra-thoracic pressure when measured out was 42 millimetres of mercury. The effect of such high pressure within the thorax is to interfere with the entrance of blood by the veins into the heart, while at the same time the abdominal viscera are rendered ischæmic by the pressure of the abdominal

muscles. The result of the first of these conditions is to diminish the amount of blood thrown out by the heart at each contraction, while the second prevents the fall of pressure which would otherwise ensue from the diminished output of the heart. We have then small pulse-waves produced with a mean arterial pressure, which in this case is somewhat higher than the normal, as can be seen from the relative heights of points half-way up the up-stroke of the earlier and later pulse-waves respectively. These points give approximately the mean

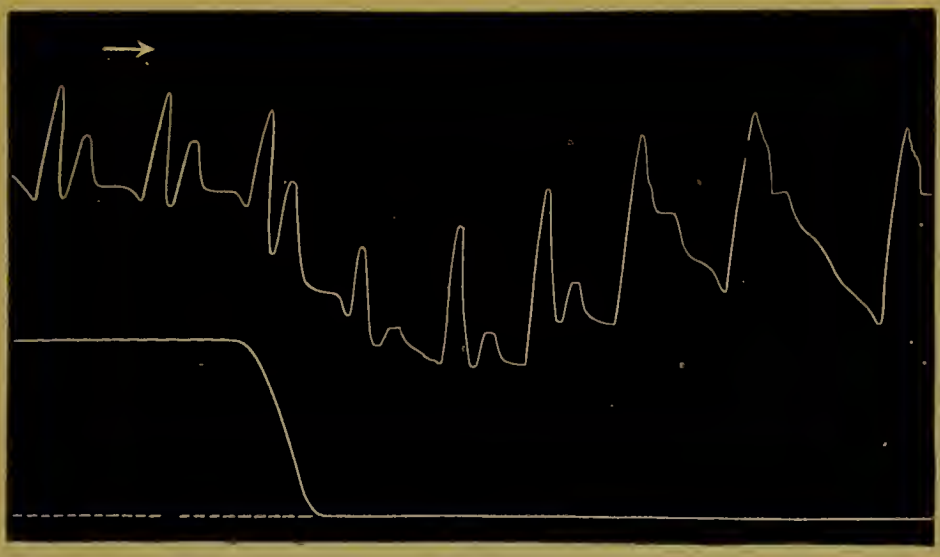

FIG. 26.

Upper curve, radial pulse obtained from healthy adult male by our modified sphygmograph (*sphygmometer*) with extra-arterial pressure of 70 mm. of mercury, showing effect of changes in intra-pulmonary and abdominal pressure. The lower uninterrupted line is recorded by a float with attached needle, resting upon the mercury of one of the limbs of a manometer, the other limb being connected with the mouth by an india-rubber tube. The lower horizontal line represents graphically the atmospheric pressure, while the upper horizontal line shows that during that time there was a positive pressure within the lungs equal to twice the distance between the two lines ($21 \times 2 = 42$ mm. of mercury).

pressure in the two cases. The first pulse-waves show greatly increased depth of the dicrotic notch, with a mean pressure above the normal. The cause of the increased dicrotism in this case is evident enough: there is only one secondary wave between the commencement of the up-stroke and the dicrotic notch; in other words, the whole of the outflow from the

ventricle takes place during the time when the increase of pressure within the heart is most rapid—that is to say, coincidently with the rapid contraction of the *musculi papillares;* and as the outflow from the heart has ceased before the commencement of the next phase of ventricular systole (when, as described on p. 93, the pressure within the ventricles is gradually falling) the termination of the outflow is very much more abrupt than is the case in the typically normal conditions. The necessary result of this is to increase the depth of the dicrotic notch, which is in this case assisted by the heightened pressure within the arterial system, causing the arterial walls to be more rigid.

To continue our analysis of this tracing: the central part shows a marked diminution of pressure, occurring as soon as the intra-abdominal and intra-thoracic pressures have been allowed to fall. As a result the abdominal veins become filled, diminishing for the moment the pressure in the systemic arteries. It can be seen that during this momentary fall the dicrotic notch, though still a marked feature, is less deep than during the first part of the curve, although the greater part if not all of the outflow still takes place during the time of active contraction of the *musculi papillares.* This, as we have pointed out, is a necessary result of the relation which exists between the arterial pressure and the elasticity of the arterial walls (p. 350).

The latter third of the curve shows the height of the pulse-waves increased to what, for the artery of this individual, is the normal height, the increased height of the waves being of course due to the increased amount of blood which is enabled to reach the heart, as the result of the filling of the abdominal and thoracic veins. With the increased output of the ventricle at each contraction, the outflow at each systole lasts a longer time, and is not confined to the phase of rapid papillary contraction (phase II., p. 93). The *outflow-remainder* secondary wave shows itself, the termination of the outflow being more gradual, and so producing no more than the normal depth of the dicrotic notch.

We may here call attention to the fact that the mechanism of the heart-beat is beautifully adapted to give the maximum rapidity of outflow with a minimum of inertia-vibrations of the column of blood in the arteries. This is due to the maximum of pressure within the ventricles being produced early in the

systole, and to the pressure being in the act of falling at the moment of termination of the outflow. The mechanism, however, only effects this economical avoidance of inertia-vibrations when the quantity of blood thrown out at each contraction, the arterial pressure, and the resistance to the escape of blood from the arteries, do not deviate from certain mean values. The above considerations lead to the conclusion that *the depth of the dicrotic notch is increased by any cause which diminishes the volume of blood which is thrown out by the ventricle at each contraction, and also by any cause which,* cæteris paribus, *raises the pressure within the systemic arteries.* We must also emphasise the fact that *a pulse-wave with greatly increased dicrotism may occur with intra-arterial pressures at or above the normal.* We shall presently have to point out that there are certain differences between the increased dicrotism of high and of low pressure. Before leaving the subject, we may note that the relation in height between the commencement of up-stroke and the dicrotic notch cannot be looked upon as a safe criterion of the height of the mean blood-pressure, as is taught by some.

Increased Dicrotism of Sthenic Fever.

By this we mean the increased dicrotism in cases of slight fever, and of more severe fevers before the circulation shows signs of failure. An example of this is shown in the lower curve of Fig. 27 (B).

The history of this curve is somewhat interesting. It was obtained, like A, from one of our own radial arteries, at a time when we were engaged every day in taking tracings from the same artery, and had been accustomed to obtain with absolute regularity a curve of the form shown in A, the pressure on the vessel being always the same—namely, that of 70 millimetres of mercury. One day the pulse-wave was found to have changed from the type of A to that of B. As the owner of the pulse felt perfectly well, we naturally at first thought the change due to something wrong with the sphygmometer, which we accordingly took to pieces: all parts of it, however, were found to be in perfect order. We took a large number of tracings on that day, all being of the type of curve B. We

finally gave up our experiments that afternoon with a feeling of bewilderment. On the following morning the explanation made itself apparent in the form of a rather severe cold in the head, which however was not so bad as to necessitate any interruption of ordinary occupations. As long as this cold lasted, and for a day or two after all other signs of it had dis-

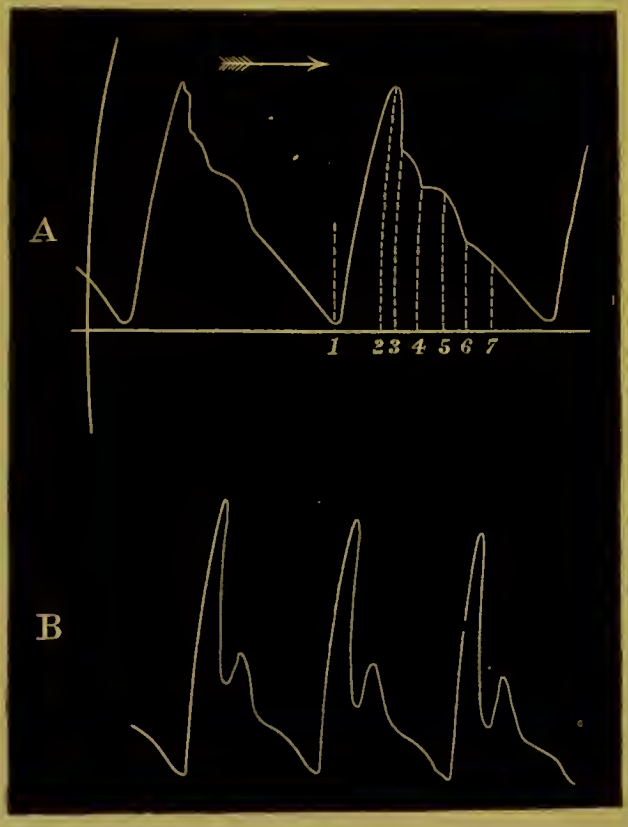

FIG. 27.

A, normal pulse from radial of healthy adult male, obtained by *sphygmometer*. Extra-arterial pressure of 70 mm. of mercury.

B, from same artery with same extra-arterial pressure, taken during acute catarrh of upper respiratory passages.

appeared, the pulse-tracings had the characters of B, gradually merging into the normal type of A.

The features of the pulse-wave may therefore in the case of a cold in the head, and presumably also in other forms of fever, become changed, and assume the characters of the febrile pulse for a good many hours before any *malaise* is felt.

With regard to the characters of this pulse it will be seen that, in spite of the increased rapidity of the heart-beat the

waves are higher than in the normal (A), indicating, so far as we know, that the amount of blood expelled at each contraction is increased, and that the amount of blood expelled by the ventricles in a given number of seconds is still more increased. In other words, the rapidity of the circulation as a whole is considerably greater than normal, and as there is no rise of the arterial pressure, indeed in all probability a fall, it necessarily follows that the resistance to the outflow of the blood from the large arteries has been diminished. Very probably in fever, although we are not aware that as yet this has been conclusively proved, the rapidity of the shortening of the myocardium is increased, in addition to the increased rapidity of beat. The curve shows that the increased volume of blood is expelled mainly during the second phase of systole (p. 93); that is to say, mainly during the time of the pulling down of the mitral valves. This, as we have pointed out in our last sub-section, increases the abruptness of the termination of the outflow, leading to the production of marked inertia-vibration, showing itself in increased depth of the dicrotic notch, or more correctly in increased height of the post-dicrotic positive wave. The great rapidity of the fall from the apex to the notch is in itself an indication of the increased freedom of outflow from the arteries.

The same form of pulse as is shown in B can be produced temporarily, that is to say, for one or two beats, by making a deep inspiration. The quantity of blood thrown out by the heart is thereby considerably increased, without increase in the peripheral resistance to the escape of blood from the arteries.

This febrile curve may pass by easy gradations into the low-pressure dicrotic curve, which we shall presently describe.

The above considerations lead to the conclusion that *diminished resistance to the outflow of blood from the arteries, by allowing the outflow to finish at an early period in the systole, may lead to marked dicrotism, even although the volume of blood expelled from the heart at each contraction is greater than normal;* although it is possible that this may in certain cases be in part due to increased rapidity of ventricular contraction. In the early stages of fever, and in mild fevers generally, these conditions are combined.

HEART-BEAT AND PULSE-WAVE.

BY C. S. ROY, M.D., F.R.S.,
Professor of Pathology,

AND J. G. ADAMI, M.A., M.B.,
Demonstrator of Pathology, in the University of Cambridge.

[From the *Cambridge Pathological Laboratory.*]

SECTION VII.—Continued.

It would in many ways be convenient were it possible from the characters of a sphygmographic curve to say at a glance what, if anything, was the matter with the person from whom it was taken. This however cannot by any means always be done, and we cannot help thinking that much of the disfavour into which sphygmographic studies have fallen is due to the fact, that the interpretation of the curves obtained is by no means so simple as is usually believed and taught. There can be no doubt that information of the greatest practical value is obtainable by the application of the graphic method to the pulse, but this can only be the case if the curves be correctly interpreted—a matter which, although often easy enough, is sometimes by no means simple on mere inspection of the tracing.

As an illustration of this we may refer to Fig. 28, taken from the radialis dext. of E. W., a female, æt. 51. This tracing at first sight seems a typically normal one, and appears to differ in no noteworthy particular from Fig. 29, which was taken from one of ourselves. And yet the first of these was obtained from a patient with facial erysipelas, whose morning and evening tem-

peratures on the day the curve was taken were 101° and 103° respectively. Are we to conclude from this tracing that the pulse of a patient on the eighth day of a severe attack of facial

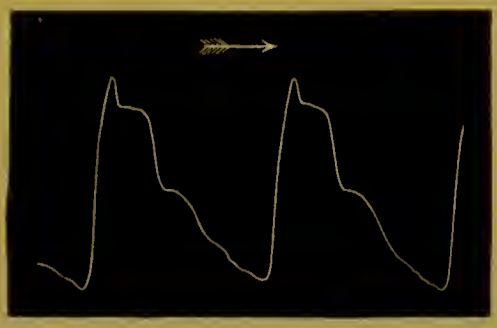

Fig. 28.

From radial artery of E. W., female, æt. 51. Suffering from facial erysipelas (eighth day). Temperature on day when tracing was taken, morning 101°, evening 103°. Pulse 65. Pressure on artery = 60 mm. of mercury.

erysipelas, with such temperatures, can show no deviation from the normal when examined by graphic methods of the extreme accuracy of those employed by us? As a matter of fact, this

Fig. 29.

From radial artery of healthy male, æt. 36. Extra-vascular pressure = 100 mm. of mercury.

woman's pulse, although of the normal form, presents a very important deviation from the normal pulse of a woman of fifty. The blood-pressure, namely, has fallen considerably. The greatest oscillations of the lever were obtained with an extra-

vascular pressure of 60 mm. of mercury, instead of 90 or 100 as would be the case with a healthy woman of this age. As we shall presently show, the maximum oscillations of the sphygmometer lever in the case of adults is obtained when the hydrostatic pressure outside the artery is a few mm. of mercury above the minimum intra-arterial pressures, *i.e.* the lowest pressures in the artery between the pulse-waves. We conclude from this observation that *the medium arterial pressure may be reduced considerably below the normal without any change in the form of the pulse-tracing being necessarily produced thereby.*

The Pulse-curve in Cases of Anæmia (Hydræmia).

We do not propose to enter here into a description of all the forms of anæmic pulse-waves, the subject being so large that a separate monograph would be required to do it justice, and we must content ourselves with giving two tracings from typically anæmic individuals.

Fig. 30 was taken from W. S., male, æt. 20, suffering from extreme anæmia, with (functional) mitral incompetence. His

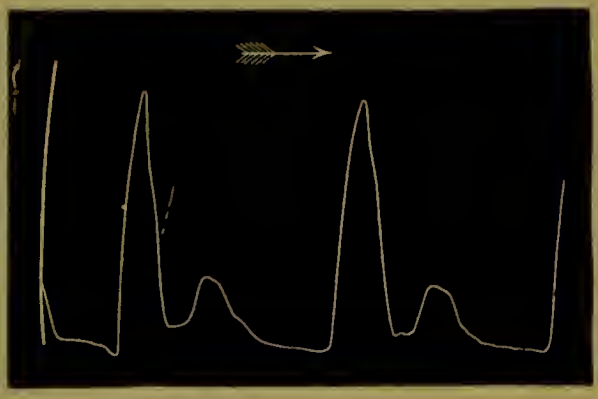

FIG. 30.

From radial of A. T., male, æt. 20. Sufferin gfrom extreme anæmia (hydræmia) with functional mitral insufficience. Extra-vascular pressure = 85 mm. of mercury.

face had a pallid yellowish tinge resembling that of pernicious anæmia; the lips were pale and waxy. There was no cyanosis or other sign of failure of the heart, beyond the mitral regurgitant murmur.

The first point to be noted about this pulse-tracing is that it shows greatly increased depth of the dicrotic notch, and would usually be described as a "low-tension" pulse. In reality, however, the arterial pressure in this youth is little, if at all, below the normal,—the minimum arterial pressure being about 85 mm. of mercury—that is to say, very much higher than in the case of the erysipelas patient whose pulse-tracing is shown in Fig. 28, and cannot be said to be below the normal for a youth of his age. On the other hand, the outflow from the heart, to judge from the characters of the tracing, lasts for a shorter time and terminates more abruptly than is normal, although the height of the up-stroke shows that the quantity of blood expelled at each systole is little if at all diminished. Presumably the abrupt termination of the outflow from the heart is the cause of the great depth of the dicrotic notch. From the pulse-tracing of this boy it can be seen that *a typically dicrotic pulse-wave of the kind usually called a low-tension pulse-curve may be present in cases where the blood-pressure in the systemic arteries is little if at all below the normal.* The nature of the heart's action has not been sufficiently taken into account as a factor in the production of increased dicrotism. A short, sharp systole with rapid propulsion of the blood into the arteries will lead to a correspondingly sharp and energetic rebound, or reactionary wave—that is to say, to increased depth of the dicrotic notch.

In Fig. 31 is given another pulse-curve from a typically anæmic person. This was obtained from J. R., male, æt. 53, whose face and mucous membranes showed the characteristic waxy pallor of great anæmia, and who also had a murmur of mitral insufficiency (functional). There was no failure of the right heart. The anæmia in his case was ascribable to ague. The highest pulse-curves were obtained with a pressure of 80 mm. of mercury, at which the tracing given in Fig. 31 was taken. The tracing shows that the amount of blood expelled at each contraction is little, if at all, below the normal. It differs, however, from the tracing in Fig. 30 in the fact that the dicrotic notch is not deeper than normal; presumably the outflow from the heart terminates less abruptly than in the case of the patient from whom Fig. 30 was obtained. The minimum blood-pressure in the case of J. R. is lower than normal, but in

spite of that his pulse-curve does not possess the characters of the so-called low-tension pulse.

We have, we believe, said enough to show that much confusion

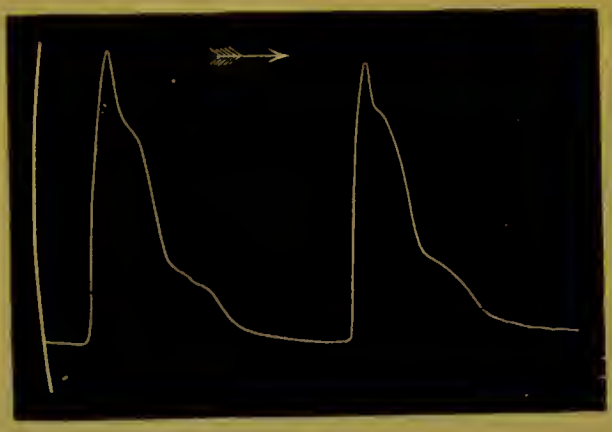

Fig. 31.

From radial artery of J. R., male, æt. 53. Suffering from anæmia and mitral regurgitation. Extra-vascular pressure = 80 mm. of mercury.

must have arisen from the ordinarily-accepted belief that the mere form of the pulse-wave is a safe guide to the height of the medium arterial pressure.

The Pulse-wave in Aortic Incompetence.

The varieties of pulse-wave which we might give are almost endless; and it is no part of our intention in this communication to give examples of all of them.

We have, for example, as yet said nothing regarding anacrotic pulse-waves, although these form a very important and fairly distinct class. What we have said, however, regarding anacrotic intra-ventricular pressure-curves applies, so far as we know, with equal force to the anacrotic pulse-wave, and we do not propose to take up the subject again here. We must also leave untouched, at least for the present, a number of other important and well-characterised forms of pulse-wave—for example, the pulses of old age and childhood, of atheroma of the arterial walls, of Bright's disease, of hypertrophy of the heart, and of the various forms of heart-disease, &c., &c. In the space at our

disposal we can only give scattered examples of the changes in the form of the pulse-wave which can be produced by various causes, and prefer to leave to others, or to a later communication, the attempt to give a complete account of the deviations from the normal which can be brought to light by the sphygmometer, or other equally accurate form of sphygmograph. We need only say here that the study of the pulse by appropriate graphic methods is one which still offers, according to our experience, an extremely rich field for investigation. We cannot pretend to have done more than dip into the subject.

Before leaving the pathological forms of pulse-wave we may give one tracing in illustration of the effect on the pulse of aortic regurgitation.

Fig. 32, from the radial of L. R., suffering from aortic regurgitation, with great hypertrophy of the left ventricle, shows

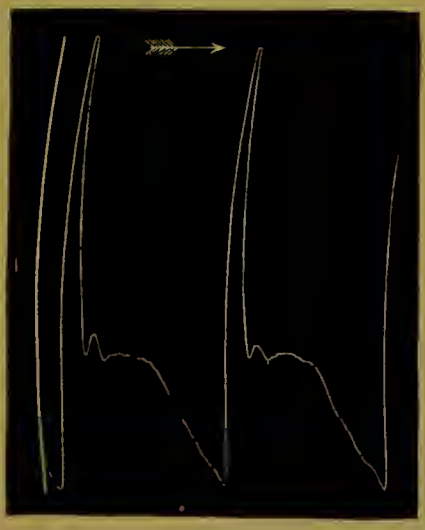

FIG. 32.

From radial of L. R., male, æt. 22. Suffering from aortic incompetence and great hypertrophy of left ventricle. Extra-arterial pressure 40 mm. of mercury.

the pulse-curve obtained with an extra-vascular pressure of 40 mm. of mercury. With 60 mm. still more ample curves were gained. One marked characteristic is the great height of the part of the tracing corresponding to the first part of the outflow from the left ventricle, as compared with the part of the curve which follows the dicrotic notch. This latter part of the tracing

indeed, is replaced by a horizontal line when the extra-vascular pressure is raised above 60 mm. of mercury. In other words, the pressure in the larger systemic arteries during the period lying between the dicrotic notch and the commencement of the next pulse-wave was very much below normal, being equal only to about 60 mm. of mercury ; while, on the other hand, the maximum pressure was very much greater than normal, 200 mm. of mercury extra-vascular pressure being required to reduce the pulse-tracing to nearly complete disappearance. The arterial walls of this individual are exposed at each heart-beat to internal pressures ranging between 60 mm. and 200 mm. of mercury. It is therefore small wonder that, in his case, all the more superficial arteries show visible pulsations.

SECTION VIII.

THE EFFECT OF VARIATIONS OF THE EXTRA-VASCULAR PRESSURE ON THE FORM OF THE PULSE-CURVE.

Fig. 20, p. 359, and Fig. 23, p. 360, show the effect on the form of the pulse-wave of variations in the pressure exerted on the portion of artery from which the tracing is taken. To illustrate this subject further, we give Fig. 33, showing the effect on an anacrotic pulse-wave of varying extra-vascular pressure ; it was obtained from the carotid of a rabbit. These three figures all show that as the extra-vascular pressure is raised from zero upwards, the curve becomes drawn out in a vertical direction, so that the secondary wave which is highest with a low extra-vascular pressure is relatively still higher as compared with the other secondary waves when the pressure is increased. As can be seen from Fig. 33, this is the case with anacrotic as well as with dicrotic pulses. We have heard it stated that with Marey's sphygmograph a pulse which is dicrotic with one pressure on the artery may be anacrotic with a different degree of pressure, and *vice versâ*. We have

not, however, met with any instance of this, and the only results of variations of the extra-vascular pressure on the form

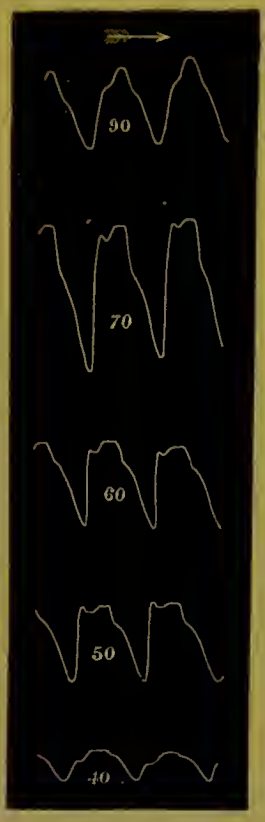

FIG. 33.

From the carotid of a rabbit, showing the effect of variations of the extra-vascular pressure on the form and height of the pulse-wave. The numbers represent the pressures on the artery in mm. of mercury.

of the pulse-wave which have come under our notice are those illustrated by Figs. 20, 23, and 33.

SECTION IX.

THE MEASUREMENT OF THE ARTERIAL PRESSURE IN MAN.

We now come to the question as to the effect of variations of the extra-vascular pressure on the height of the pulse-curves, and to the very important question of the absolute measurement of the arterial blood-pressure in man.

Attempts have been made, as need perhaps hardly be said, to graduate the pressure of the spring of Marey's sphygmograph so as to obtain some idea of the degree of compressibility of the pulse, and so to obtain some information, necessarily vague at the best, of the arterial pressure. The results obtained by this means have, however, been so little satisfactory that it is employed, so far as we know, by very few at the present time.

We are not aware that anyone has seriously sought to show that trustworthy information regarding the absolute arterial pressure in man can be obtained by Marey's sphygmograph. It has been sought to measure the arterial tension in man without employment of the graphic method by Von Basch and by Potain.

The principle of Von Basch's sphygmomanometer is to compress the artery by a cushion, whose interior is connected with a column of mercury, the height of which can be varied at will, and which oscillates with each pulse-wave until the pressure is raised sufficiently high to prevent the pulse-waves entering the compressed portion of artery. There are, we believe, two serious objections to this method, viz. first, the fact that to make the pulse-beats evident, a long column of mercury requires to be set in motion, and the inertia of this necessarily proves a serious source of error; and, secondly, the fact that at the best the method can only give information regarding the maximum intra-arterial pressure. We doubt much whether any confidence can be placed on the results obtained by its use.

Potain also compresses the artery by means of a cushion, the pressure in which is known, but avoids the employment of the column of mercury to give information regarding the moment of occlusion of the artery, which he determines by means of the finger placed on the vessel on the distal aspect of the cushion. The objections to this method are obvious, and we need only mention that the recognition of the moment of occlusion of the artery depends on the tactile sensibility of the physician's finger, the accuracy of which we do not believe is so great as that of an appropriate recording-instrument. There is, moreover, the risk of the pulse-wave being conveyed to the part of the artery lying beyond the cushion by means of the comparatively free anastomosis through the palmar arch. Besides these objections the fact that only the maximum pressure in the artery can be

measured by this method is of itself, we think, a fatal objection to it.

We should not have considered it necessary to criticise these two methods of measuring the blood-pressure in man were we not convinced that in the sphygmometer we possess the means of measuring with accuracy both the minimum and the maximum pulse-pressures, *i.e.* the pressures both at the bases and at the apices of the pulse-waves, and also with approximate accuracy the pressure at any part (*e.g.* at the level of the dicrotic notch) of the pulse-wave. It need hardly be said that both the maximum and the minimum pulse-pressures are influenced by the respirations, so that the true maxima and minima of the arterial pressure are the pressures of the apices of the pulse-waves at the summits of the respiratory rises, and of the bases of the pulse-waves at the bases of the respiratory oscillations of the arterial tension. It will simplify matters considerably, however, if we leave the effect of the respiratory movements on the arterial pressure out of consideration in the meantime. When therefore in the following pages we refer to the maximum and minimum arterial pressures, it must be understood that we refer to these pressures as measured during temporary cessation of the respiration with the glottis open.

In order that what we have to say on the subject may be understood, it is necessary to give some account of the instrument which we have employed, and which we will refer to as the sphygmometer. We had hoped that by this time we should be in a position to give a description of the final form, so far as we are concerned, of this instrument. As, however, its details are still being modified and improved, we must content ourselves in the meantime with a description of the principle on which it is constructed, leaving a detailed description of it to be published in a few weeks' time.

The diagram, Fig. 34, shows its salient characters. It consists of a box (*a*) which is moulded below so as to fit the anterior surface of the lower end of the radius, bridging over the point of entrance and exit of the radial artery. In this box is a flexible bag, shown in dotted outline at *b*, filled with water, and connected by a T-tube with a rubber bag and a mercury manometer. By means of the tap (*c*)

the fluid in the box after having been raised to any desired manometric pressure can be cut off from the bag and the

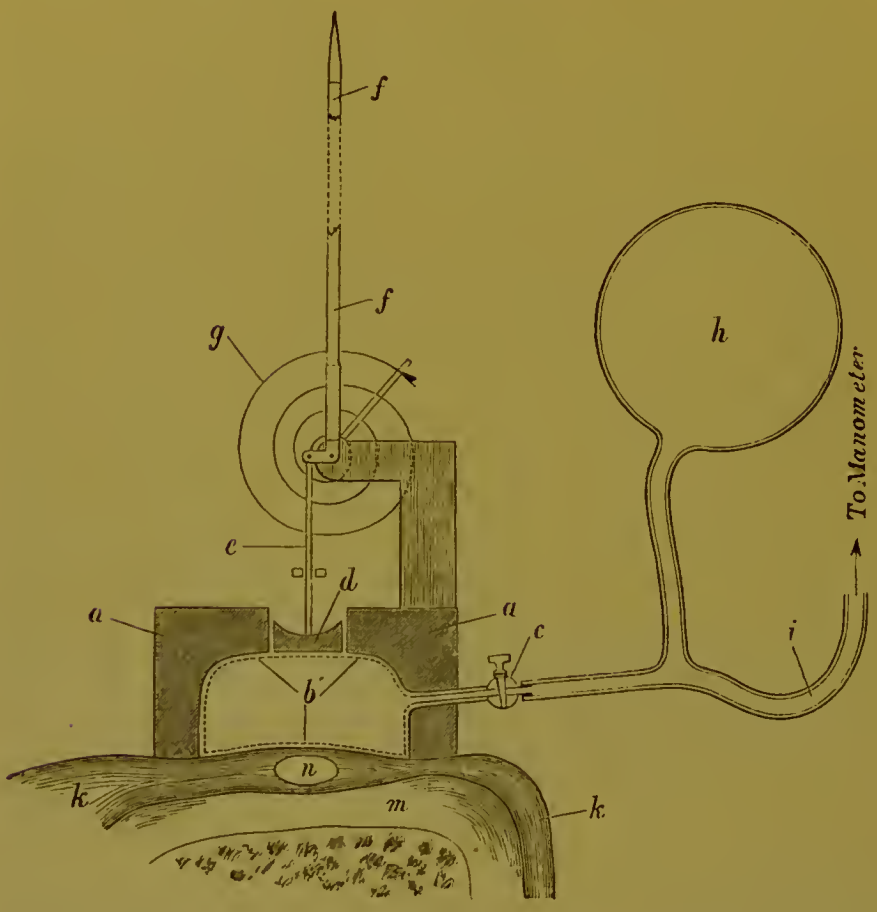

FIG. 34.

Diagrammatic sectional representation of sphygmometer.

a. Box in which portion of artery is enclosed.
b. Thin-walled rubber bag filled with water and communicating through tap (*c*) with manometer and thick-walled rubber bag (*h*).
d. Piston connected by rod (*e*) with recording lever (*f*).
g. Spiral spring attached to axis of lever, and by which the pressure in *b* against the piston (*d*) is counterbalanced.
k. Skin and subcutaneous tissue.
m. End of radius seen in section.
n. Radial artery seen in section.

manometer. At the upper part of the box is a circular opening, in which, and resting on the bag (*b*), works a flat button (*d*), connected to the lever by a short light rod (*e*), whose upper end hinges on a horizontal projection from the axis of the vertical

lever (*f*). Attached to the axis of rotation of this lever is the spiral watch-spring (*g*), which can be tightened at will, so that the lever can be made to assume a vertical position at any desired hydrostatic pressure within the box. Finally, by means of an appropriate mechanism a piece of blackened glazed paper is made to move in a vertical direction past the end of the recording lever.

In actual use the box is first fixed firmly over the end of the radius by an appropriate holder, and the tap (*c*) being opened, the pressure within the box is raised to any desired height, the spring being tightened or slackened until the lever is vertical: then, the tap having been closed, tracings can be obtained without difficulty. The pressure within the box acts in all directions, and is correctly given by the manometer.

Let us now see how with an instrument of this kind the height of the pulse-wave is affected by changes in the pressure exerted upon the artery. The diagram, Fig. 35, illustrates this in the case of a healthy male, *æt. circa* 40. In this diagram the abscissæ give the manometric pressures upon the artery, while the ordinates give the heights of the pulse-wave as recorded by the sphygmometer. It can be seen that with a pressure of 20 mm. of mercury the pulse-wave is low, and that its height rises in a rapidly increasing ratio upon the addition of each 20 mm. of pressure, until at a pressure of 100 mm. of mercury it attains its maximum. At pressures above this it falls rapidly, tending to describe a curve which runs asymptotically with the abscissa line. It must be understood that this curve varies with different individuals, and at different ages. As however this subject is being worked out accurately by Mr. L. Rolleston, at our suggestion, we need not here enter further into the subject, except that we must interpret the meaning of this characteristic curve. Why, first of all, is the pulse-curve highest when the extra-arterial pressure is so high as in this case, 100 mm. of mercury? Why, in other words, do the contents of the portion of artery in the box undergo the greatest oscillations in volume when the extra-arterial pressure is in this instance at this particular height? The explanation, we believe, is not difficult.

Let it be clearly understood, in the first place, that the height

of the pulse-waves as recorded by an instrument of this kind depends entirely on the extent of the variations of the cubic contents of the piece of artery within the box, which result from the changes in the intra-arterial pressure at each pulse-beat. They are not directly influenced, as is the case for example with a plethysmograph enclosing the hand, by the amount of blood which passes a given point of the arterial tube in a given time.

Let us assume, for the sake of illustration, that the pulse-waves are in a given case measured in height by 30 mm. of mercury,

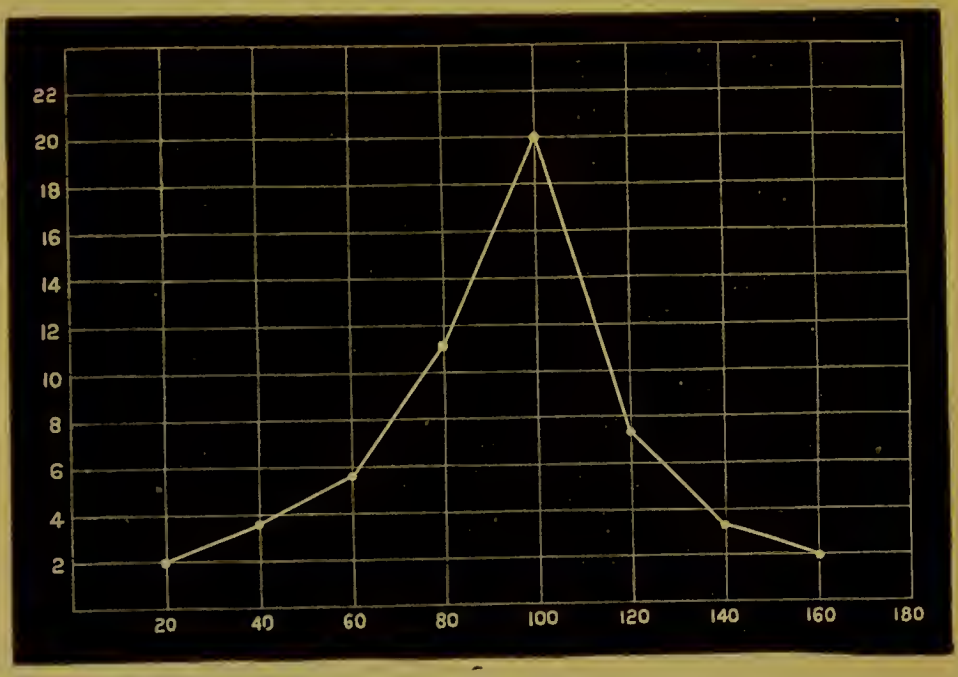

FIG. 35.

Curve of height of sphygmometer pulse-waves with different extra-arterial pressures. From healthy male, æt. 40. The abscissæ are the extra-vascular pressures in mm. of mercury. The ordinates are the heights of the pulse-waves.

and let us now consider what will be the mean arterial pressure at which waves of this height will cause the greatest alterations of the cubic contents of a portion of one of the larger arteries—in other words, what mean pressure will give the largest oscillations of the sphygmometer lever.

It must be kept in mind that, as has been shown by one of us,[1]

[1] Roy, "Elasticity of the Arterial Wall," *Journ. of Physiology*, vol. iii. (1881).

the arteries of adults in man are relatively wider in the undistended condition (*i.e.* when the intra- and extra-vascular pressures are equal), so that when the intra-vascular pressure is reduced but very slightly below that outside the vessel, or when the extra-vascular pressure is very slightly raised above the intra-vascular pressure, a relatively great change in the cubic capacity of the artery results, owing to the flexible-walled tube undergoing collapse. Knowing this fact, it is not difficult to see that a pulse-wave of 30 mm. of mercury in height will produce the greatest change in the cubic contents of a portion of artery if at each pulse-wave the artery is alternately collapsed and opened out.

It need hardly be said that, in normal conditions, the pressure in the arteries never sinks below that outside the vessel walls—never, in other words, becomes sub-atmospheric. The same effect can be produced, however, in so far as the relation between the intra-vascular pressure and the cubic contents of any given piece of artery is concerned, by raising the extra-vascular pressure. For example, a pulse-wave which causes a rise of pressure within the artery from 100 to 130 mm. of mercury will, if the extra-vascular pressure be raised to 105 mm. of mercury, produce the same maximum change in the cubic contents of that portion of artery as if the intra-arterial pressure oscillated between -5 and $+25$ mm. of mercury, with the extra-arterial pressure equal to that of the atmosphere.

We would expect, therefore, the pulse-waves recorded by the sphygmometer to reach their maximum size when the extra-vascular pressure reaches a height which just exceeds[1] the minimum intra-vascular pressure.

Let us now see how, in individual cases, the size of the pulse-wave is affected by the extra-vascular pressure. The simplest way to do this is probably to construct a curve the abscissæ of which will represent the extra-vascular pressures, while the ordinates show the corresponding heights of the pulse-waves. Fig. 35, as we have already said, is such a curve, taken from a healthy male æt. 40.

[1] The pressure required to cause collapse of a portion of an artery like the radial, after excision, is so small that it may safely be ignored in the present connexion.

We see then that by means of the sphygmometer it is possible within small limits to find what is the minimum arterial pressure. With regard to the maximum arterial pressure, this, so far as we can learn, lies at that point of the abscissa-line which is cut by a continuation of the descending line of these curves, as is shown by the interrupted lines (*a*) in Fig. 35.

So far as our observations on this subject have carried us, we find that both maximum and minimum arterial pressures, as well as the distance of these two apart, vary with the age, sex, and condition, as well as with disease of the individual. The subject is being worked out by Mr. L. Rolleston.

In conclusion, we must express our regret that our papers on the heart-beat and pulse-wave should have run to such an inordinate length, and still more that in spite of our having written so much we have been obliged to omit for the present a great deal that we consider of practical or theoretical importance.